The Effect of Rapid Eye Movements on Imaging in EMDR and Treatment of PTSD.

This thesis contains no material which has been accepted for the award of any other higher degree or graduate diploma in any university. To the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except when due reference is made in the text of the thesis.

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A Review of the Treatment Components and Effectiveness of EMDR in PTSD.

By

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Abstract

Post Traumatic Stress Disorder (PTSD) is treated psychologically with exposure and cognitive restructuring techniques. Shapiro's (1995) Eye Movement Desensitisation and Reprocessing (EMDR) treatment for PTSD involves imaginal exposure, cognitive restructuring, *and rapid eye movements* (EMs). EMDR has been presented as an advance in the treatment of PTSD, based on the theory that rapid EMs facilitate the accelerated processing of trauma-related information, via the activation of physiological mechanisms which stimulate retrieval of positive memories and emotions (Shapiro, 1995). It is suggested in this literature review that Shapiro's EMs theories are unsound, and that claims that EMDR is superior to traditional PTSD treatments are premature, but it is proposed that EMDR may have therapeutic benefits in treating PTSD. It is suggested that the exposure and cognitive restructuring components of EMDR may be more critical than the EMs in reducing PTSD symptoms. However, the EMs may facilitate client acceptance of these components by distracting clients from their anxiety and reducing the intensity of imaginal exposure. Alternative theories of the role of eye movements in EMDR need to be investigated in future research.

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Literature Review

Eye Movement Desensitisation and Reprocessing (EMDR) (Shapiro, 1995) is a new treatment for Post Traumatic Stress Disorder (PTSD), combining imaginal exposure to traumatic memories, cognitive restructuring of negative self assessments, and the performance of rapid back-and-forth horizontal eye movements (EMs). Psychological treatments for PTSD are traditionally long term and involve exposure, anxiety management, and cognitive restructuring techniques. The eye movements in EMDR, however, are proposed to provide immediate relief of PTSD symptoms and long term benefits. These claims, coupled with mixed findings from empirical investigations of the efficacy of EMDR, have placed EMDR at the centre of much controversy. This investigation will address research into EMDR and Eye Movement Desensitisation (EMD), which was the original, less refined version of Shapiro's treatment (Shapiro, 1989).

Definition of PTSD

PTSD is a disorder resulting from the experience of an extreme stressor. According to the American Psychological Association criteria for PTSD (APA, 1994) a person with PTSD must have had a direct personal experience of an event that contained actual or threatened death or serious physical harm, or must have witnessed such an event happen to someone else. Response to this event must have involved intense fear, helplessness, or horror. The necessary symptoms for a PTSD diagnosis involve the person with PTSD persistently re-experiencing the trauma via intrusive thoughts, dreams, and flashbacks; persistently avoiding stimuli associated with the trauma and having numbed general responsiveness;

and/or having persistently increased arousal. These symptoms must cause clinically significant distress and impairment in social, occupational, or other important areas of functioning, and must be present for more than one month.

The promise of EMDR

EMDR treats PTSD because clients are asked to examine and alter any irrational beliefs they may have in a safe environment where the clinician's presence and the eye movements are cues or reminders of the client's present day reality - that the trauma is over. Clients become desensitised to their traumatic memories by combining imaginal exposure of the memories with rapid eye movements. EMDR is proposed to result in a lasting reduction of anxiety, changes in the cognitive assessment of the memory, and cessation of flashbacks, intrusive thoughts, and sleep disturbances (Shapiro, 1989). During an EMDR session the client may recall memories, experience new emotions, and gain insights into his or her negative beliefs which are causing distress. The client may experience an abreaction, in which he or she becomes very disturbed and re-experiences the emotional intensity of the trauma. Shapiro suggests that this will lead to new insights and may reveal more pertinent negative beliefs. Finally, the trauma image may change to a neutral image or memory, with the client no longer being able to retrieve the original image, and anxiety may change to calm.

When first testing EMD with trauma victims, Shapiro (1995, p 4) reported that 'Doug', a Vietnam war veteran, found that his most traumatic war image was transformed to look like "a paint chip under water" and that his anxiety had changed to calm, as he rapidly moved his eyes. At six months follow up

'Doug' could only retrieve his 'paint chip' image, and when asked to think of Vietnam he now remembered it as a "garden paradise".

EMDR procedures for the treatment of PTSD

Shapiro outlines eight stages of EMDR therapy as follows:

1) **History taking and treatment planning** procedures in EMDR involve the therapist's assessment of the client's current mental and physical ability to withstand intense emotion. Pregnant clients and those with respiratory or cardiac conditions are excluded. Caution is required with epileptic clients, and safety factors must be instituted for clients with suicidal ideation, impoverished support networks, eye problems, and substance abuse problems. The therapist then assesses the clinical picture and establishes the treatment goals.

2) In the **preparation phase** a therapeutic alliance is established. The therapist explains EMDR, enhances the client's expectancy of success, and discusses the possibility of emotional disturbance. The client is then taught relaxation techniques to deal with emotional disturbance during and after imaginal exposure to the trauma.

3) During the **assessment phase** the client identifies an **image** of the target trauma memory, as the whole incident or its most disturbing aspect. In EMDR every detail of the trauma and response must be targeted, via imagination, during a visual tracking task. The client must identify the **negative beliefs** which are causing inappropriate, dysfunctional behaviour and affect. Negative beliefs are generally current self-assessments held by the client, such as the belief 'I am powerless'. Next, the client formulates a preferred, realistic **positive belief**. This belief is given a rating by the client on a scale from one 'completely false' to

seven 'completely true' (Validity of Cognition scale, Shapiro, 1989), which reflects the client's 'gut response' to the statement. Effective positive beliefs are 'I' statements that incorporate an internal locus of control, such as 'I am lovable' rather than 'he will love me', and which can generalise over the broadest range of dysfunctional material. The client also focuses on his or her **current emotions**, which are elicited when picturing the image and rehearsing the negative belief, and then rates his or her level of disturbance from zero 'neutral intensity' to ten 'highest possible anxiety', on a Subjective Units of Distress (SUDs) scale (Wolpe, 1982).

4) During the **desensitisation phase** the client's negative trauma-related affect and beliefs are 'reprocessed', which involves concentration on the target affect or belief whilst performing sets of eye movements (EMs). A typical EMs set involves the clinician holding two fingers upright, palm facing the client, approximately 12 to 14 inches from the client's face. Full bilateral eye movement is elicited by asking the client to track the therapist's horizontally moving fingers at the maximum comfortable sustainable speed (Shapiro, 1995). The finger sweeps are generally one left to right, and back to left, movement per second, across a horizontal distance of at least 12 inches (Shapiro, 1989). This elicits a combination of smooth pursuit EMs (continuous visual tracking of a moving object) and saccadic EMs (very brief, high velocity EMs up to 600°/second), when the therapist's hand is moving faster than the eye is able to accurately track (Armstrong & Vaughan, 1996). EMs sets are 12-to-24, or 24-to-36, bilateral movements and are continued until the client's Subjective Units of Distress score indicates little or no distress. The direction, speed, and distance of the clinician's

finger movements can be varied for client comfort, and to facilitate a change in the client's response.

As an alternative to EMs, the clinician may tap each of the client's upturned palms, with his fingers, rapidly alternating left and right taps. Also the clinician can snap his fingers next to each ear of the client alternately, at a comparable speed to that used with the EMs sets (Shapiro, 1995).

5) The negative belief is considered to be replaced by the positive belief in the **installation phase**. The EMs sets are repeated while the client focuses on the positive belief and the target memory. This continues until the client's 'gut response' to the positive belief is a rating of six or seven on the Validity of Cognition scale. The aim is to empower the client, and to permanently link the trauma memory with a positive self interpretation.

6) In the **body scan phase** of EMDR the client focuses on the fully installed positive belief and the target memory, and scans his or her body for **physical sensations** associated with the trauma memory. These physical sensations are then reprocessed with EMs.

7) In the **closure phase** of EMDR the client is debriefed. The therapist instructs the client to keep a journal of negative thoughts and to continue using relaxation techniques.

8) Finally, **re-evaluation** refers to the process of assessing the maintenance of positive effects at the beginning of each new session. The client's journal is reviewed for new target material, or, alternatively, previously treated trauma memories may need to be reprocessed.

The treatment components of EMDR, and the role of EMs

Shapiro (1995) proposes that the following procedural components of EMDR, *in addition to the EMs*, account for its therapeutic effectiveness (Shapiro, 1995):

1) Clients are helped to repeatedly create and dismiss their traumatic imagery. They are also asked to 'just notice' the physical sensations, emotions and thoughts, which are induced when imagining their trauma. This objectivity, combined with the safety cues of the clinical context, may increase the client's sense of mastery over the trauma memories, and reduce the client's anxiety.

2) The client's verbal identification of his or her negative self assessments may highlight their irrationality to the client. Likewise, the cognitive restructuring inherent in formulating positive beliefs may facilitate recovery. The procedural reconnection of the trauma material may help the client to make sense of the experience, facilitating its storage in narrative memory where it can be used for personal growth.

3) After thorough preparation, the client is repeatedly exposed to the image, which opposes the avoidance reaction that maintains PTSD.

It has been alternatively concluded that the EMs component is not necessary, because the procedural components that Shapiro describes are simply good exposure therapy and cognitive therapy (Andrade, Kavanagh, & Baddely, 1997; Allen & Lewis, 1996). Likewise, it has been suggested that the component parts of EMDR, *minus the eye movements*, equal good psychotherapy (Hyer & Brandsma, 1997). The non EMs components of EMDR are good psychotherapy because it involves investigation of the unique associative networks created by the

individual, to protect the self from trauma. Also the therapist remains non-directive, allowing the client to be inwardly focussed, the specifier of therapy targets, and to feel self empowered. Moreover, the therapist takes on the principle that the client will naturally move toward positive growth given the right environment, and enhances the client's treatment expectations (Hyer & Brandsma, 1997). Shapiro argues, however, that the EMs are necessary for the rapid effects of EMDR (Shapiro, 1998), because the EMs facilitate *accelerated information processing.*

Shapiro's theories regarding the necessity of EMs in EMDR

Shapiro (1989) has formulated a physiological based theory of EMDR, in which EMs are considered to activate an innate, self healing, information processing system. Shapiro suggests that this system may become unbalanced by traumatic events or unresolved stress, causing PTSD. This is based on Pavlov's (1927) hypothesis that traumatic incidents upset the excitatory/ inhibitory neural balance in the brain, causing a pathological change in the neural elements. This in turn is considered to block the usual progression of information processing to resolution, maintaining the trauma in active memory and thus causing intrusive thoughts, flashbacks, and nightmares (Shapiro, 1989). It is also proposed that rapid eye movements, rather than exposure or cognitive restructuring, unblock the information processing system at an accelerated rate. This proposal is explained by the following model and related theories:

The Accelerated Information Processing (AIP) Model

In the Accelerated Information Processing model, a memory is conceptualised as a target node with connected neurophysiological network channels, which contain associated beliefs, emotions and physical sensations. The EMs in EMDR are proposed to result in the connection of neuro-network channels containing positive information to trauma memory nodes. Once adaptive information is incorporated into the associated network channels of trauma memory nodes, which Shapiro calls 'reprocessing', the trauma memory shifts from non-declarative to declarative (narrative) memory storage. At this stage the reprocessed memory can be used for personal growth. Shapiro (1995) states that the dysfunctional material connected to a trauma memory node can be reprocessed at an accelerated rate, because EMDR has a generalised positive influence throughout the neurophysiological network, and memories can be targeted in clusters. Thus Shapiro proposes that pathologies can be cured in a limited period of time, making EMDR an advance in PTSD treatment.

Shapiro (1995) outlines numerous, mainly untested, theories as to how EMs may facilitate information processing, and listed below are three of these theories: First, she considers that the EMs and alternative stimuli may activate unknown physiological mechanisms or brain functions. This may disrupt the client's complex, habitual, physiological responses to the traumatic memory and allow information processing to occur (Shapiro, 1995). Second, Shapiro proposes that repetitive EMs may elicit neural bursts, causing a low voltage current which decreases the synaptic potential of all neural networks. Networks containing trauma information are proposed to have very resistant receptors, and so are

unable to link with positive content networks. However, it is hypothesised that the progressive lowering of receptor resistances results in linkages between negative and positive content networks. Thus traumatic memories are reprocessed because they are linked to self enhancing knowledge. This explains reported observations of clients' progressive discharge of negative affect, the evolution of more adaptive beliefs, and the recall of positive memories, across EMs sets (Shapiro, 1995). Finally, Shapiro considers that the EMs may induce a relaxation response, which may in turn facilitate information processing. EMs may cause changes in the reticular formation of the brain stem or mechanisms that activate the parasympathetic nervous system, inducing relaxation (Shapiro, 1995). The parasympathetic nervous system is activated during tasks that demand visual convergence (Monnier, 1968), perhaps explaining EMDR treatment effects in studies which have substituted EMs with eye fixation (Dunn, Schwartz, Hatfield, & Wiegele, 1996; Pitman et al., 1993; Renfrey & Spates, 1994).

To explain the effects of EMs to clients, Shapiro (1989) compares the EMs in EMDR to rapid eye movement (REM) sleep (Shapiro, 1995, p 121). REM sleep is postulated to be involved in memory and stress-related information processing, and people with PTSD have altered REM sleep (Gabel, 1987; Winson, 1993). These reports have lead to Shapiro's hypotheses that eye movements are associated with information processing and anxiety.

However, the EMs/REM comparison simply draws attention to an overt similarity between Shapiro's EMs and REM, without showing that the underlying mechanisms of REM sleep are identical to those of EMDR (Page & Crino, 1993). Also, REM sleep may serve a physiological information processing function, but

the rapid eye movements during REM sleep may represent only an epiphenomenon of dreaming. Thus, these rapid eye movements may result from, rather than induce, information processing (Pitman, et al., 1996a).

The above theories suggest that the EMs are an essential PTSD treatment component. However, the speculation that the positive treatment effects of EMDR are due to the exposure and cognitive restructuring components of EMDR, and are not, as Shapiro suggests, due to the EMs, is fuelled by 1) criticisms of Shapiro's theories, and 2) evidence for the effectiveness of exposure and cognitive restructuring techniques in treating PTSD.

Criticisms of Shapiro's theories

Shapiro's acknowledgment of the therapeutic effects of EMDR procedural components, other than the EMs, is arguably her only straightforward theorising and the credibility of EMDR is claimed to be undermined by Shapiro's "sketchy neurobiological theorising" (Allen & Lewis, 1996, p 250). Shapiro credits EMDR with amazingly rapid effects, which she explains by stating that conventional therapies use verbal procedures to process information; whereas EMDR involves physiological based procedures (Shapiro, 1995, p 46). However, it is argued that performing EMs is no more inherently "physiological" or "neurobiological" than speaking, listening, or thinking (Allen & Lewis, 1996, p 250). Shapiro has also been criticised about the claim that EMDR has rapid effects, her insistence that this has a neurophysiological explanation, but her amendment that the associated physiological mechanism is not yet known or understood (Shapiro 1995, p 53 & p 310) lends EMDR an "arcane quality" (Allen

& Lewis, 1996, p 251). Moreover, the language Shapiro (1995) uses, such as saying that it is necessary to "clean out" (p 33) the channels of traumatic memory networks, and that positive beliefs are incorporated into memory networks via the "installation" process (p 157), promotes the misconception that the client is passive while his or her neurophysiology is being mysteriously altered by EMs (Allen & Lewis, 1996, p 251). Also, the explanation of the Accelerated Information Processing model in terms of "neural balance" and "neural pathology" ambiguously refers to "unidentified effects of unidentified processes on unidentified neural substrates" (Page & Crino, 1993, p 292). Finally, there is limited evidence to support Shapiro's theories that eye movements facilitate information processing via physiological mechanisms.

A critique of the theory that EMs induce relaxation

The theory that eye movements induce relaxation and are an essential component of EMDR has had limited support (Hedstrom, 1991, Wilson, Silver, Covi, & Foster, 1996). The EMs are compared to hatha yoga eye exercises (Hedstrom, 1991), which are proposed to distract attention from personal problems, decrease muscular tension, and increase relaxation and concentration (Dechanet, 1965; Satchidananda, 1970). Increased relaxation is explained in terms of an increased production of alpha waves when the eyes are closed or defocused, even if the participant is engaged in mental activity and attention (Hedstrom, 1991). This explanation can be applied to EMDR because 1) the alpha wave state would decrease clients' arousal when thinking about their trauma, 2) alpha production is closely tied to the visual areas of the brain and so

may be triggered by EMs, and 3) EMDR clients' eyes are likely to defocus trying to maintain the rapid tracking motion (Sharpley, Montgomery, & Scalzo, 1996). However, it has been shown that relaxation and alpha wave activity is not elicited by EMs (Sharpley, et al., 1996). This finding is explained by the contention that the original comparison between Shapiro's EMs and hatha yoga eye exercises is superficial (Page & Crino, 1993).

Alternatively it has been demonstrated that psychophysiological measures of relaxation indicate that EMDR clients experience a 'compelled relaxation response' (Wilson, et al., 1996). A study of six participants with PTSD found that EMD caused a consistent, but not significant, reduction in heart rate and blood pressure (Montgomery & Ayllon, 1994a). Moreover, heart rate and systolic blood pressure were significantly decreased during EMD in a single PTSD case study (Montgomery & Ayllon, 1994b). Also, EMDR treatment of PTSD has been shown to significantly decrease participants' between session electromyogram levels (Forbes, Creamer, & Rycroft, 1994). However, other researchers have been unable to replicate reduction of physiological arousal during EMs (Boudewyns, Stwerka, Hyer, Albrecht, & Sperr, 1993; Carlson, Rusnak, Chemtob, & Hedlund, 1996; Sharpley, et al., 1996), or during post-EMDR *in vivo* exposure (Foley & Spates, 1995).

Some authors have noted reductions in traumatised clients' arousal levels, due to PTSD treatments *not* involving EMs. In a study comparing routine clinical care, EMDR, and biofeedback-assisted relaxation training, in treating combat related PTSD, clients' psychophysiological measures reflected a habituation effect from pre- to post- treatment, and were not differentially effected

by treatment condition (Carlson, Chemtob, Rusnak, Hedlund, & Muraoka, 1998). One study even found that four sets of 24 finger taps significantly reduced participants' heart rates more than EMD (Merckelbach, Hogervost, & Kampman, 1994). Therefore, demand and expectancy effects, and procedural elements of EMD/EMDR other than EMs, may be responsible for clients' 'relaxation responses', a clinical observation which has not been confirmed in controlled research.

A comparison of the treatment effects of exposure, cognitive restructuring, and EMs

Imaginal and *in vivo* exposure to trauma material is considered to be beneficial because avoidance maintains fear, whereas confrontation of trauma stimuli can lead to new insights and an improved response to trauma stimuli. In 'flooding' the client remains in the feared situation until his arousal subsides, and cognitive change is promoted by re-exposure to the trauma memories without additional injury and loss. In 'systematic desensitisation', the person learns to associate relaxation with each part of the fearful situation. Cognitive therapies, likewise, help clients to see the irrationality and self destructive nature of their fears and negative beliefs, and to formulate alternative beliefs which are self enhancing.

The successful use of exposure to treat PTSD has been observed in controlled studies examining systematic desensitisation (Brom, Kleber, & Defares, 1989; Peniston, 1986), imaginal exposure and flooding (Boudewyns & Hyer, 1990; Cooper & Clum, 1989; Foa, Rothbaum, Riggs, Murdock, & Walsh, 1991;

Keane, Fairbank, Caddell, & Zimering, 1989), with positive results being maintained for two years (Peniston, 1986), and intrusive symptoms being consistently improved. These studies measured significant reductions of nightmares, flashbacks, muscle tensions, hospital readmissions (Peniston, 1986); reductions in fear, state anxiety, depression (Keane, et al., 1989); and in sleep disturbances and "psychotic-like" symptoms (Cooper & Clum, 1989). Likewise, success in treating PTSD with 'stress inoculation therapy', a cognitive therapy, has been reported (Foa et al., 1991). It has been concluded however, that prolonged exposure is more effective than stress inoculation therapy in reducing the intrusive symptoms of PTSD (Foa et al., 1994), and so exposure may be a more important treatment component than cognitive restructuring.

Flooding is sometimes an inappropriate treatment for PTSD, however, because it may elicit too much anxiety and precipitate a panic disorder, and flooding may only address anxiety symptoms, with its usefulness in treating guilt, anger, emotional numbing, irrational beliefs, and shame unclear (DeBell & Jones, 1997; Solomon, Gerrity, & Muff, 1992). Likewise, systematic desensitisation can be problematic in treating PTSD, because of the difficulty in constructing a hierarchy of the least-to-most distressing components of rape and war. Also, systematic desensitisation requires that the client's anxiety levels remain very low with only gradual increments, which is difficult to achieve because high levels of disturbance are associated with PTSD (Spector & Huthwaite, 1993). Finally, exposure based treatments, in particular flooding, are not 'treatment of choice' for some clients because they do not wish to 'relive' their trauma (Scott & Stradling, 1997; Stern & Marks, 1973).

This author is not aware of any studies which directly compare the therapeutic benefits of EMDR with flooding, systematic desensitisation, or stress inoculation therapy in treating PTSD. However, based on a comparison of EMDR and an eye fixation control treatment, it has been concluded that imaginal exposure to trauma material, combined with rehearsal of positive beliefs, is an effective treatment of PTSD, with EMs being non-essential (Renfrey & Spates, 1994). The EMs condition in this study, however, appeared to take significantly less time to achieve treatment effects than the eye fixation condition (Shapiro, 1996). Likewise, a comparison of the effects of EMDR and an EMDR analogue with eye fixation, in treating chronic combat related PTSD, has also failed to support the necessity of EMs (Pitman, et al., 1996a). Both treatments produced modest to moderate improvement on standardised measures of PTSD, with slightly more improvement in the eyes-fixed condition than the EMs condition. However, the authors concluded that EMDR was at least as efficacious for combat related PTSD as was imaginal flooding in a previous study, and that EMDR was better tolerated by participants (Pitman, Altman, Longpre, Poire, & Macklin, 1996b). These conclusions were considered to be invalid however, because the treatments were not directly compared in a single controlled study, and the two studies were not comparable (Cahill & Frueh, 1997). Thus, the inclusion of EMs in treating anxiety disorders has not consistently improved upon the treatment outcomes produced by including exposure and cognitive restructuring techniques. However the inclusion of EMs may facilitate the effectiveness of the exposure and cognitive restructuring components of EMDR, by making these treatment components more tolerable. EMDR may, thus, have a clinical role when

traditional exposure therapies have failed or are inappropriate because clients are too anxious or avoiding to cope with a standard exposure treatment (Andrade, et al., 1997).

EMs make EMDR a user-friendly exposure therapy

It may be considered that the EMs in EMDR may decrease the intensity and adverseness of imaginal exposure by reminding clients that they are safe, distracting client's from their anxiety, and increasing clients' treatment expectations. These considerations are supported by the following three theories: 1) It is proposed that the EMs in EMDR reduce the vividness and emotiveness of traumatic imagery. This may in turn reduce the client's anxiety response to being exposed to trauma-related stimuli, allowing habituation to the traumatic memory to occur via the other EMDR procedural elements. This hypothesis is based on the observation that people cannot simultaneously perform two visuospatial tasks, and so when people attempt to hold a personally emotive image in their mind whilst performing rapid EMs, or a pattern tapping task, the vividness and emotiveness of the image is reduced (Andrade, et al., 1997). This is attributed to a disruption of the functioning of the Visual Spatial Sketchpad (VSSP) of working memory, by the dual visuospatial tasks. The VSSP is suggested to process visual and spatial information and to have limited capacity. The VSSP combines with the phonological loop - which processes auditory information, and these two systems are coordinated by the central executive system in Baddeley's (1986) model of working memory. It is proposed that rapid EMs interfere with

concurrent short term memory of visual information, by competing for processing resources (Andrade, et al., 1997).

2) Dyck (1993) suggests that the EMs are a distraction from the trauma memory, allowing patients to remain relaxed, and extinguishing the anxiety response which would generally follow reminders of the trauma. In the 'conditioning' model of PTSD, traumatic incidents are associated with an anxiety response which disrupts subsequent learning other than avoidance or escape learning. It is suggested that in EMDR the traumatic memory is repeatedly paired with no anxiety, due to the distraction effects of EMs, and that this leads to extinction of the anxiety response and so allows learning to occur. It is also suggested that when the client is not sufficiently distracted by the EMs, he or she experiences intense anxiety (abreaction), and a flooding paradigm may explain the treatment effects (Dyck, 1993).

3) Alternatively, the EMs may not fully distract the client from the trauma, but may serve to remind the client of his present safety as he focuses on the trauma, extinguishing his anxiety (Armstrong & Vaughan, 1996). This theory is explained by the Orienting Response model of EMD/EMDR. The orienting response (OR) is a human behavioural reflex, which actively extracts important survival information from the environment. It is triggered by environmental novelty, or by significant stimuli. Components of the OR can include bodily movements, increased sensory perception, autonomic changes, de-synchronisation of alpha rhythm, and visual search via saccadic (lateral) EMs (Armstrong & Vaughan, 1996). An investigatory search which does not identify danger leads to de-arousal, and an enabling of approach behaviour (McCulloch & Feldman, 1996).

The EMD/EMDR client may be primed for ORs in the presence of trauma-related stimuli. When instructed to focus on the memory, the client's physiological arousal increases. The waving hand is given significance, due to task instructions and the context of the memory, and so facilitates an intense OR. The ensuing investigation of the environment establishes the client's safety. Thus, de-arousal is repeatedly paired with the trauma memory, replacing the conditioned emotional response and inhibiting avoidance behaviour (McCulloch & Feldman, 1996). The OR extinguishes rapidly to non-significant innocuous stimuli, and more slowly to significant stimuli. If stimulation is continued after extinction then a tonic inhibitory state associated with drowsiness is reported to develop (Sokolov, 1963).

Theories that EMDR treatment effects are due to clinician elicited ORs and distraction effects of clinician elicited EMs may explain reports that EMDR treatment effects have limited generalisation to non-clinical settings (Sanderson & Carpenter, 1992), that minimally distracting cues such as eye fixation produce minimal therapeutic effects (Montgomery & Ayllon, 1994a), and reports that therapists and patients prefer this procedure over more direct exposure procedures (Boudewyns & Hyer, 1996, p192). However there have also been reports of participants finding EMD/EMDR procedures too aversive (Lipke & Botkin, 1992; Marquis, 1991; Oswalt, Anderson, Hagstrom, & Berkowitz, 1993), and so the above theories that EMs make EMDR a user-friendly exposure therapy need to be further investigated.

Evidence for and against EMD/EMDR treatment for PTSD

More than ten thousand clinicians were trained in EMDR by 1995, under the EMDR Institute (Shapiro, 1995). This enthusiasm for EMDR may have

originated from early EMD/EMDR case studies, which had some very positive results, and Shapiro's (1995) finding that there were more controlled studies supporting the efficacy of EMD/EMDR in treating PTSD, than for any other psychological PTSD treatment. However, many of the early studies on EMD/EMDR did not have rigorous scientific controls, and controlled research on traditional PTSD treatments is limited (Solomon, et al., 1992).

Case reports

Published case reports have generally had mixed or supportive findings for the use of EMD/EMDR in treating PTSD. However these findings cannot be generalised to all PTSD participants, are subject to publishing bias as new therapies are more likely to be published if reports are positive (Page & Crino, 1993), and are confounded by procedural and design flaws. In most of these reports the results are non-conclusive because of over-reliance on the client's self reports and therapist's impressions, failures to formally diagnose symptoms or to establish the baseline stability of symptoms, and the uncontrolled effects of prior and concurrent therapies (Kleinknecht & Morgan, 1992; Marquis, 1991; McCann, 1992; Oswalt, et al., 1993; Puk, 1991; Shapiro, 1989; Spates & Burnette, 1995; Spector & Huthwaite, 1993; Wolpe & Abrams, 1991).

One positive case study reported that EMD improved non-combat related PTSD symptoms, for eight out of ten participants, after only one to four sessions (Vaughan, Weiss, Gold, & Tarrier, 1994). Failure to have successfully treat two of the participants may be attributed to insufficient treatment time. However, the positive results were also confounded because two participants did not yet have

the PTSD diagnosis, three participants were concurrently taking medication, and changes were not statistically reliable (Lohr, Kleinknecht, Tolin, & Barrett, 1995).

In a study by Forbes, Creamer, & Rycroft (1994) which contained few procedural flaws, it was concluded that four sessions of EMDR may be at least moderately effective in reducing civilian and combat related PTSD symptoms. However, following treatment, significant pathology remained and four of the eight participants still met the criteria for a full diagnosis of PTSD.

Finally, it has been suggested that numerous sessions of EMDR are needed to treat PTSD sufferers with multiple or complex traumas, such as war veterans. In a case study of four Vietnam war veterans with PTSD, it was found that 12 sessions of EMDR resulted in substantial clinical improvements for three of the veterans on standardised measures of cognitive-behavioural symptoms of PTSD (Carlson, et al., 1996).

Empirical evidence supporting the use of EMD/EMDR in treating PTSD

It was initially reported that one session of EMD produced substantial desensitisation and cognitive restructuring of trauma related perceptions, with results being maintained at three months follow up (Shapiro, 1989). However, participants only received eight minutes of placebo treatment before EMD - calling into question the face validity of the placebo, the amount of exposure was not standardised across treatments, there was no waiting list control, and there may have been experimenter bias and expectancy effects. Also demand effects may have confounded results, because the EMD condition was only terminated upon significant improvement in the Subjective Units of Distress scale and

Validity of Cognition scale ratings, and EMD was applied for 15 to 90 minutes. Data was limited to the Validity of Cognition and Subjective Units of Distress ratings, however the Validity of Cognition scale is not validated and may assess affective lability more than irrational processes making it "redundant" (Lohr et al., 1992, p 163).

An improvement on Shapiro's (1989) study showed that three 90 minute EMDR sessions decreased the anxiety and PTSD symptoms, and increased the positive beliefs, of 80 participants with a traumatic memory, 46 percent of whom had PTSD (Wilson, Becker, & Tinker, 1995). Participants who were randomly assigned to a delayed treatment condition showed no improvement until administered EMDR. Effects were maintained after 90 days (Wilson et al., 1995) and 15 months (Wilson, Becker, & Tinker, 1997). However, comorbidity and behavioural measures of outcome were not assessed, and the trauma specific symptoms were improved more than general psychological functioning.

A study of rape victims with PTSD revealed that three 90 minute sessions of EMDR were effective in alleviating PTSD symptomatology, compared to a waitlist control (Rothbaum, 1997). The EMDR treatment had good fidelity and 10 percent of the EMDR group met full criteria for PTSD at post treatment, compared to 88 percent of participants in the control condition. However, there were no significant differences between the groups on measures of general fear and anxiety or dissociation at post treatment. The author suggested that the treatment effects of EMDR were comparable to the cognitive behavioural treatments studied by Foa et al. (1991), but admitted that such conclusions must

be drawn from controlled comparisons between EMDR and the other validated PTSD treatments.

The effects of EMDR in treating non-combat related PTSD were compared to the effects of a standard care treatment for civilian PTSD, and it was concluded that both treatments have a positive impact on symptoms (Marcus, Marquis, & Sakai, 1997). However, EMDR participants demonstrated significantly greater and more rapid improvement on inventories for PTSD, depression, and anxiety. Likewise, two sessions of EMDR significantly improved the post test measures of PTSD, depression, and anxiety, for traumatised young women (Scheck, Schaeffer, & Gillette, 1998). This outcome was significantly greater than the positive effects afforded by an Active Listening Treatment, in which the therapist used attentive silence and non-judgemental acknowledgment of the participants' communication (Scheck, et al., 1998).

A comparison of the relative effects of the incremental addition of EMDR, relaxation training, and biofeedback, to usual milieu treatment for Vietnam war veterans, showed that EMDR was generally the most effective extra treatment (Silver, Brooks, & Obenchain, 1995). However this study had procedural flaws, neither biofeedback or relaxation are validated as effective treatments for PTSD (Solomon, et al., 1992), and the authors' conclusions have been discredited as resulting from an improper statistical analysis (Lohr et al., 1995).

Finally, a recent study compared the effects of 12 sessions of EMDR, 12 sessions of biofeedback assisted relaxation, and routine clinical care, on the treatment of combat related PTSD (Carlson, et al., 1998). Compared with the

other treatments, EMDR resulted in significantly greater improvements on cognitive-behavioural measures of PTSD at post test, three months follow up, and nine months follow up.

Empirical evidence opposing the use of EMD/EMDR in treating PTSD

Some empirical research has suggested that EMD/EMDR should not be chosen above traditional therapies in the treatment of PTSD. In a comparison of EMD, image habituation training and applied muscle relaxation training, no significant difference was found between the treatments, which all reduced PTSD symptoms and depression (Vaughan, Armstrong, et al., 1994). Thus, because image habituation training and relaxation have not been validated as treatments for PTSD (Solomon, et al., 1992), EMD was only as effective as a credible placebo (Lohr et al., 1995).

In a comparison of two 90 minute sessions of EMDR, two 90 minute exposure control (no EMs) sessions, and a no treatment control, for combat veterans who were also receiving standard milieu treatment, no significant differences between the groups on standardised PTSD measures were demonstrated, and only within session Subjective Units of Distress ratings decreased for EMDR participants (Boudewyns, et al., 1993). This supports the argument that EMDR effects are limited to decreasing within session anxiety (Lohr et al., 1995). However this study also raises the problems of treating combat related PTSD with only two sessions of EMDR and veterans over-reporting symptoms for compensation benefits. If measures of general psychological functioning are used with multiple event trauma, participants should have at least 12 weeks of EMDR (Shapiro, 1995, p 325).

A second study has also shown that EMDR decreases war veterans' Subjective Units of Distress scores, but that at post treatment there are no differences between these veterans and those only receiving standard milieu treatment, on Validity of Cognition ratings and standard measures of symptomatology (Jensen, 1994). However, this study suffers the same flaws as Boudewyns et al. (1993).

The current status of EMDR compared to traditional PTSD treatments

EMDR has been accepted as an empirically validated treatment and 'probably efficacious for civilian PTSD' by a Task Force of the Clinical Division of the American Psychological Association (APA, 1998), based on the findings of Wilson, et al. (1995) and Rothbaum (1997). Thus EMDR has joined the status of exposure therapy and stress inoculation therapy. This status, requires that two studies indicate that the treatment is more efficacious than a waiting list control.

However, compared to traditional exposure-based psychological PTSD treatments, EMDR has been proposed to be more effective, more efficient (although complex cases require at least 12 sessions), and better tolerated by clients and therapists (Pitman et al., 1996a; Wilson, et al., 1997; Hassard, 1993; Macculloch & Feldman, 1996; Hyer & Brandsma, 1997; Carlson et al., 1996). Shapiro argues that there is sufficient evidence to hail EMDR as an advance in PTSD treatments (Grant, 1998) and that the APA Task Force should increase EMDR's status to 'well established for PTSD *in general*' (Shapiro, 1998). This is based on her criticisms that equivocal results with combat populations are due to insufficient treatment time and fidelity, and failures to address all traumas in

complex PTSD (Jenson, 1994; Boudewyns et al., 1993), and on recent findings which support the use of EMDR with combat-related and civilian PTSD (Scheck, et al., 1998; Carlson, et al., 1998).

Claims that EMDR is a superior PTSD treatment, compared to traditional exposure and cognitive restructuring therapies are unwarranted. EMDR still needs to be directly compared to systematic desensitisation and flooding. Also the negative findings of EMDR effectiveness can not be simply attributed to an absence of EMDR training or treatment fidelity, as treatment effects should be robust enough to withstand procedural modification (Dr Kavanagh, in Grant, 1998; Lohr et al., 1995).

Conclusion

It is concluded that EMDR is an effective treatment for civilian and combat related PTSD when all details of clients' traumas are addressed over a number of sessions. However, arguments that EMDR is superior to traditional PTSD treatments are premature, due to a lack of controlled comparisons of EMDR and validated PTSD treatments. It is instead suggested that EMs simply facilitate client acceptance of conventional cognitive restructuring and exposure, which are components of EMDR. The EMs may achieve this by distracting clients from their anxiety and reminding clients of their safe environment, via an intense orienting response. Thus EMDR may help clients who have found traditional exposure based treatments, like flooding, too aversive. More research is needed to uncover the role of EMs in EMDR.

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An Assessment of the Effect of Rapid Eye Movement on Imaging in EMDR and Treatment of PTSD.

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Journal Article

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Appendices

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An Assessment of the Effect of Rapid Eye Movement on Imaging in EMDR and Treatment of PTSD.

Abstract

Eye Movement Desensitisation and Reprocessing (EMDR) is a treatment for Post Traumatic Stress Disorder (PTSD) which involves the client visualising traumatic memories, whilst performing rapid eye movements (EMs). The developer of EMDR, Shapiro (1995) proposes that EMs facilitate accelerated information processing and induce relaxation. The aim of the present research was to measure imaging ability and eye movements in four conditions. There were two baseline tasks called **Baseline Imaging** and **Baseline EMs**, and two experimental tasks called **Imaging + EMs** and **Imaging + Fixation**. Participants were a group with PTSD and a matched control group. The imaging task was adapted from Logie, Zucco, and Baddely (1990) and involved participants listening to aural instructions for mentally constructing and visualising 3 X 5 matrices of black and white squares which formed number or letter characters.

The eye movement conditions - the **Baseline EMs** and **Imaging + EMs** tasks, involved participants tracking a target spot, which moved back and forth at a speed of 44 °/second and across a distance of 20 °. The EMs elicited were similar to those in EMDR. These EMs were performed during completion of the imaging task in the Imaging + EMs condition. The **Imaging + Fixation** task involved the measurement of imaging ability whilst fixating on a target spot and during which another spot moved horizontally across the screen at a speed of 44 °/second and across a distance of 20 °. During each stage of the experiment, participants' average heart rates, perceptions of task difficulty, and subjective anxiety were measured, to investigate whether the EMs induced relaxation.

The results showed that there were no significant differences between the PTSD and matched control group in eye movement and imaging ability. The eye movement analysis showed that the EMs were predominantly smooth pursuit eye movement with a small saccadic eye movement component. The results showed that, compared to baseline imaging ability, there was a significant decrease in imaging ability when participants had to simultaneously fixate their gaze on a stationary target. Likewise, compared to imaging ability during eye fixation, there was a significant decrease in imaging ability when participants had to simultaneously perform EMs. Analyses of subjective anxiety, perceptions of task difficulty, and average heart rates showed that the EMs induced anxiety and increased the difficulty of imaging, when they were presented as a secondary task. It is concluded that EMs may reduce the vividness of traumatic images, by disrupting visualisation, and thus distract clients from their anxiety in EMDR.

Eye Movement Desensitisation and Reprocessing (EMDR, Shapiro, 1995) is a combination of imaginal exposure, cognitive restructuring, and psycho-dynamic principles for a client-centred protocol, with the addition of rapid eye movements. EMDR is mainly associated with the treatment of civilian and combat related Post Traumatic Stress Disorder (PTSD), and has recently had some impressive results (Marcus, Marquis, & Sakai, 1997; Scheck, Schaeffer, & Gillette, 1998; Carlson, Rusnak, Chemtob, & Hedlund, 1996; Carlson, Chemtob, Rusnak, Hedlund, & Muraoka, 1998). EMDR has received support as a 'probably efficacious' treatment for civilian PTSD, by a task force of the Clinical Division of the American Psychological Association (APA, 1998). This support was based on the findings of Wilson, Becker, and Tinker (1995) and Rothbaum (1997), which indicated that EMDR is a more efficacious PTSD treatment than a waiting list control. EMDR is considered to induce lasting reductions of anxiety and PTSD symptoms (up to 15 months according to Wilson, Becker, and Tinker, 1997), changes in cognitive assessment of traumatic memories with improved self appraisal, and cessation of flashbacks, intrusive thoughts, and sleep disturbances (Shapiro, 1989).

EMDR treatment for PTSD involves the client first identifying his or her trauma-related beliefs, emotions, bodily sensations, and memories. This is followed by the client being asked to imagine the distressing trauma details, whilst he or she concurrently performs sets of 12-24 or 24-36 bilateral rapid eye movements (EMs). To initiate eye movements the clinician holds two fingers upright, palm facing the client, and instructs the client to track these fingers as they are moved horizontally in front of the client's eyes. The EMs are performed at the maximum comfortable speed, generally one back and forth movement per second, across a distance of at least 12 inches (Shapiro, 1989). There are variations as to how these EMs may be elicited, or

they can be replaced completely with rapidly presented bilateral tactile or auditory stimuli (Shapiro, 1995, p 67). The EMs sets are repeated until the visualisation of each distressing detail of the trauma has become significantly less anxiety provoking, according to the client's self rating on a Subjective Units of Distress (SUDs) scale (Wolpe, 1982). At this point the client is considered to be desensitised to trauma-related stimuli. The client is then asked to focus on a self enhancing belief, which he or she has identified as desirable, again whilst performing sets of rapid eye movements. These EMs sets are repeated until the client believes the positive statement, as rated on a Validity of Cognition (VOC) scale (Shapiro, 1989).

The EMs component is based on Shapiro's chance observation, whilst walking in a park one day in 1987, that her disturbing thoughts were suddenly disappearing and were not returning without conscious effort. Shapiro scrutinised her actions and noticed that her disturbing thoughts were coupled with spontaneous, rapid back-and-forth eye movements in an upward diagonal direction. Shapiro found that deliberately making the eye movements had the same effect of reducing the negative charge of her thoughts. She deduced that the eye movements were saccadic (very brief, high velocity eye movements, up to 600°/second, Armstrong & Vaughan, 1996), and formulated the initial version of her therapy, Eye Movement Desensitisation (EMD). EMD only differs from EMDR in terms of a reduced emphasis on bodily tension and less refined protocols.

Shapiro's description of the origin of EMD has been questioned on the premise that normal saccadic eye movements appear to be physiologically undetectable, and are typically triggered by external stimuli (Rosen, 1995). However, it has been argued that Rosen's conclusion is erroneous due to an incomplete understanding of saccadic eye movements and humans' ability to sense them, and that

it is most likely that Shapiro's spontaneous eye movements were indeed saccadic (Welch, 1996). The EMs elicited in EMD and EMDR are thought to be smooth pursuit eye movements (the ocular motor system moves the eye smoothly and continuously at a rate that ideally matches that of the moving target) with some saccadic eye movements (occasional saccades bring the fovea on to the target to correct errors of eye position in relation to the moving target), when the therapist's hand is moving faster than the eye is able to track (Armstrong & Vaughan, 1996). There are several proposals to explain why repetitive, saccadic eye movements spontaneously accompanied Shapiro's negative thoughts, and how eye movements facilitate the treatment effects of EMDR.

It is claimed that the EMs cause the proposed treatment effects of EMDR by inducing both a 'compelled relaxation response' and accelerated processing of trauma-related memories, emotions, and beliefs, to achieve resolution (Shapiro, 1995; Hedstrom, 1991; Wilson, Silver, Covi, & Foster, 1996). These effects are considered to be manifested in clients' rapidly changing emotions, cognitive insights, and recalled memories, across single sessions of EMDR (Shapiro, 1995). The purpose of this study is to investigate the effectiveness of the eye movement component of EMDR in facilitating relaxation, and information processing in terms of the ability to visualise an image or event.

Based on observations that psychophysiological arousal is reduced during EMDR, it is suggested that the EMs induce a 'conditioned' or 'compelled' relaxation response, by activating unknown physiological mechanisms which in turn activate the parasympathetic nervous system (Shapiro, 1995; Wilson et al., 1995). Therefore, by asking the client to visualise and imagine the trauma whilst performing the EMs, the traumatic memory or scene becomes associated with relaxation, instead of anxiety.

Similarly, Marquis (1991, p 192) suggested that field currents generated by the EMs "interfere with tracts connecting the frontal lobes with the hypothalamus and hippocampus in such a way as to weaken the connection between stimulus and response", causing a de-conditioning of the client's anxiety response to trauma-related stimuli.

Shapiro (1995) also explains her proposal that the EMs facilitate accelerated information processing by arguing that the EMs cause neural bursts. These neural bursts add up to a low voltage current, which decreases the synaptic resistances of hypothetical neuro-networks. These neuro-networks store associated memories, affect, and information. The synaptic potential of the various neuro-networks increases according to the intensity of affect stored within. High valance networks therefore contain traumatic memories and anxiety, and cannot link up with low valance networks, which contain positive memories and adaptive information. However, linkage may occur between these networks when the proposed low voltage current, generated by the EMs, decreases the synaptic potential of negative content networks. Thus, negative memories and images are connected to self enhancing beliefs, and so are 'reprocessed'. Finally, it is also proposed that positive effects generalise across neuro-networks and that trauma memories can be targeted in clusters, and so the processing of emotional and cognitive information is accelerated.

The necessity of including the EMs in EMDR, and the ability of the eye movement component to facilitate trauma desensitisation and accelerated information reprocessing, has been questioned. Numerous studies have failed to show that eye movements induce relaxation, either during the performance of EMs (Boudewyns, Stwerka, Hyer, Albrecht, & Sperr, 1993; Carlson, et al., 1996; Sharpley, Montgomery, & Scalzo, 1996), or during post-EMDR *in vivo* exposure (Foley & Spates, 1995).

Likewise, reductions in traumatised clients' arousal levels due to PTSD treatments *not* involving EMs have been reported (Carlson, et al., 1998; Dunn, Schwartz, Hatfield, & Wiegele, 1996; Renfrey & Spates, 1994; Merckelback, Hogervost, & Kampman, 1994). It has been shown that the exclusion of the EMs, or alternative stimuli, in EMDR, does not significantly effect treatment results (Renfrey & Spates, 1994; Sanderson & Carpenter, 1992; Pitman, et al., 1996). These findings, that the EMs are a non-essential component of EMDR, support arguments that imaginal exposure to trauma stimuli and rehearsal of self-enhancing beliefs are the critical treatment components of EMDR. Likewise, these findings contradict theories which attribute a critical physiological role to the EMs in the reprocessing of trauma information. Moreover, the ease with which the EMs are effectively replaced by auditory stimuli (Cocco & Sharpe, 1993) and a finger tapping task (Bauman & Melnyk, 1994), suggests that the EMs do not play a critical physiological role in trauma desensitisation, unless Shapiro's (1995) claim that the EMs are not unique in their physiological effects is accepted.

The theory that the EMs facilitate the processing of emotions, and reduce the emotional impact of traumatic situations, has not received experimental support. It has been demonstrated that emotional processing of an aversive photograph is significantly impaired when non-clinical participants are given rapid EMD (rapid EMs were two EMs per second, as suggested by Shapiro, 1995) (Tallis & Smith, 1993). These participants continued to report 'definite' distress, in response to the photograph, after 20 blocks of saccades. This impairment was not shown by participants given either slow EMD (slow EMs were one eye movement per second), or a repeated imaginal exposure treatment with no EMs, who reported only 'slight' distress after equal treatment time. This study replaced Shapiro's finger tracking

EMs, which participants reported to *interfere with their ability to maintain focus on a mental image*, with a task where participants tracked shoulder taps with their eyes closed.

Another study has demonstrated that EMD does not reduce non-clinical participants' emotional re-activity to an aversive photograph, any more than a placebo treatment (Merckelbach, et al., 1994). This placebo treatment involved the repeated tapping of the participant's right index finger during imagery exposure. The authors concluded that EMD treatment effects can be attributed to the imaginal exposure component, and to placebo, demand, and/or expectancy mechanisms (Merckelbach, et al., 1994). However, it has been observed that consistent decreases in the emotiveness of traumatic imagery, which is visualised whilst performing rapid EMs, only occurs when participants imagine traumatic *personal* recollections (Andrade, Baddely, Kavanagh, & Baddeley, 1997).

Failures to demonstrate that EMs facilitate emotional processing may be explained by the observation that people report more visual imagery when their concentration is interrupted following ocular quiescence (no EMs greater than 3°), than when it is interrupted following spontaneous eye movement (Antrobus, Antrobus, & Singer, 1964). Rapid EMs may interrupt visualisation, in turn inhibiting the emotional processing of imagery. The observation that a participant had no spontaneous eye movements whilst imagining a distressing scene, but performed spontaneous eye movements to interrupt these thoughts, is anecdotal evidence that performing EMs interrupts the visualisation of aversive imagery (Antrobus et al., 1964). The authors suggest that people attempt to 'break up' thoughts that they wish to suppress by changing their visual input by making eye movements.

The theory that EMs facilitate cognitive processing has similarly been debated. Associations between eye movements, emotions and cognitive processes have been reported, which support Shapiro's proposals. A lateral eye shift has been observed which appears to be related to the shifting of attention and to be reduced or abolished when anxiety is high (Day, 1964). Additionally, it has been reported that mental concentration is not associated with ocular fixation on a single object, but with directed gaze with rhythmic ocular movements, which are hypothesised to correlate with beta-wave activity and "greater cerebral activity" (Teitelbaum, 1954, p 354).

However, it may be considered that saccadic eye movements are negatively correlated to cognitive processing. This is because participants' spontaneous saccadic eye movements have been observed to decrease, whereas the duration of their ocular quiescence increases, during their completion of an increasingly difficult auditory discrimination task, which involves increasing demands for short term memory (Antrobus, 1973). Similarly, the range of extent of saccadic eye movements has been observed to decrease significantly as mental workload increases in tone counting and visual counting tasks (May, Kennedy, Williams, Dunlap, & Brannan, 1990). Practise effects do not seem to prevent reductions in saccadic amplitude, even when participants' performances improve, so long as the task remains difficult (May et al., 1990).

The suppression of cognitive processing (specifically mental rotation) during saccadic eye movements has also been directly observed. When participants were 1) shown an orientation prime during eye fixation which provided information about the orientation of the stimulus to be identified, 2) then were asked to move their eyes across a short or a long saccade, before 3) being shown the rotated number or letter character they had to identify, they were not advantaged by having a long saccade,

compared to a short saccade, separate the prime and target stimuli (Irwin & Carlson-Radvansky, 1996). However, the time difference between long and short saccades has been shown to be effective, in improving participants' response times, in a non-saccade control condition (Irwin & Carlson-Radvansky, 1996).

Finally, it has been suggested that eye movements are correlated to participants' rate of cognitive change (Antrobus, et al., 1964). A correlation between eye movements and rapid cognitive change may explain the proposed increase in cognitive and emotional processing during EMDR. Participants may not be able to concentrate on a single thought and so report changing beliefs, emotions, and memories.

It may be that saccadic eye movements aid in concentration and visualisation. This may be because saccadic eye movements suppress sensitivity to visual input. Attention to external visual information during the processing of trauma information would exhaust the client's limited span of attention. However, these saccadic eye movements may also suppress cognitive activity, and this may be no more noticeable than the suppression of visual input that accompanies saccades (Irwin & Carlson-Radvansky, 1996). It has not been investigated what kinds of eye movements are generated by the moving finger stimulus in EMDR. Research is needed to show whether involuntary and voluntary eye movements facilitate cognitive processing, and to define which types of eye movements are associated with which emotions and which cognitive processes.

Accordingly, the present research will investigate the kind of eye movements that are generated during EMDR treatment, and whether these EMs have an effect on visual cognitive processing and anxiety as suggested by Shapiro (1995). The design of the research involves the measurement of baseline eye movements in response to

instructions to track a moving red light, in a Baseline EMs task, and visual imaging ability across three tasks: (1) a baseline measurement of imaging ability called Baseline Imaging, (2) imaging ability in an eye fixed condition where the participant stared at a stationary red light with another red light moving in front of the stationary eyes, called Imaging + Fixation, and (3) imaging ability with eye movement or tracking of the moving light, called Imaging + EMs. Participants belong to either a group with PTSD or a matched control group. In each of the tasks (Baseline EMs, Baseline Imaging, Imaging + Fixation, Imaging + EMs) average heart rate will also be measured, as well as self-reports of anxiety and difficulty of the tasks. Based on the proposal that eye movement will interfere with visual cognitive processing, it is predicted that in comparison to Baseline Imaging there will be a reduction in imaging ability in the Imaging + EMs task. It is also proposed that in comparison to the Baseline Imaging task there will be a reduction in imaging ability in the Imaging + Fixation task, however this reduction is not predicted to be as significant as in the Imaging + EMs task. Finally, it is predicted that there may be a 'compelled relaxation response' in the Baseline EMs and Imaging + EMs tasks, based on Shapiro (1995). The present study will also compare the eye movements in the Baseline EMs task with the eye movements in the Imaging + EMs task, and it is expected that there will be no significant differences between the eye movements elicited in these two tasks.

Method

Participants

Participants in the PTSD Group were respondents to newspaper advertisements and community notices. The kind and level of their symptoms of

mental disorder fulfilled DSM-IV criteria for a PTSD diagnosis (American Psychological Association, 1994). (See Appendices 1 and 2 for newspaper advertisement and poster). Participants in the PTSD Group completed the Posttraumatic Stress Diagnostic Scale (Foa, 1995), which assesses PTSD symptoms according to DSM IV criteria (APA, 1994), and provides ratings of 'level of impairment of functioning' and 'severity of symptoms'. Participants in the PTSD Group also completed the Impact of Events Scale (Revised) (Weiss, 1993), which assesses participants' level of distress concerning PTSD symptoms over the past week.

One of the PTSD Group participants had a 'moderate-to-severe' level of impairment of functioning, and 12 had a 'severe' level of impairment of functioning. One participant had a 'moderate' symptom severity rating, five participants had 'moderate-to-severe' symptom severity, and seven participants had 'severe' symptom severity. Each PTSD Group participant's distress due to intrusion of trauma-related thoughts, avoidance of trauma-related stimuli, and/or hyper-arousal, over the previous seven days, was clinically significant. Examples of traumas cited as the source of participants' PTSD symptoms included personal experience of sexual and/or physical abuse by a stranger and/or family member, combat and torture in a war zone, serious car accident, life-threatening illness, finding a brother murdered, and a forced abortion at the age of 14 years, and witnessing a stabbing, a father drown, and a partner overdose on illegal drugs.

Control participants responded to advertisement within the University of Tasmania, and were matched to participants in the PTSD group on age, sex, years of education, and score on the Vocabulary scale of the Weschler Adult Intelligence

Scale-Revised (WAIS-R, 1981). Control participants had no traumatic memories or PTSD symptoms.

Participants in the PTSD and Control Groups completed the Symptom Checklist 90-Revised scale (SCL 90-R, Derogatis, 1993). The SCL 90-R contains measures of somatisation, obsessive-compulsive behaviour, interpersonal-sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism, and provides a global severity index (GSI), a positive symptom distress index (PSDI), and a positive symptom total (PST). Likewise, both Groups completed the Vocabulary scale of the WAIS-R (1981), to check that no participant had impaired cognitive functioning.

There were 13 participants in each of the Control and PTSD Groups. The Control Group had 8 females, and the PTSD Group had 7 females. No participant had a concurrent psychotic disorder or a substance abuse problem, as determined by the Symptom Checklist-90-Revised (Derogatis, 1993) and an unstructured clinical interview. One participant in the PTSD Group was currently taking 50 mg Zoloft per day, and another was taking 200 mg Zolfoft per day for depression.

Apparatus

The taped instructions, for the imaging task, were presented using head phones. Participants' average heart rates were recorded during each stage of the experiment, on a Bioview Series IV biofeedback unit, attached to a PC computer, with a pulse sensor that clipped on to the participant's earlobe.

Measurement of Eye Movements

The eye movement system was controlled by a host PC computer. The target stimulus was generated by a red back-propagated laser beam controlled by a General

Scanning Model XY0507V X-Y Optical Scanning Head (General Scanning Inc.), used to deflect a Uniphase Helium Gas laser beam onto the back of a large stimulus screen. The scanning head was controlled by a Model DSC2005 Series Digit Scan Controller (General Scanning Inc.). The diameter of the red laser spot was 0.15° of visual angle, and its luminance was reduced to 0.5 cd/m² using a Wratten No.2 filter, on a background of 0.2 cd/m². The contrast of this target spot was calculated using Michelson's formula for contrast (Lmax - Lmin / Lmax + Lmin) and was 0.4. The target spot was back-propagated on a large stimulus screen, which was a REARLITE (Opra) opaque projection screen with a horizontal dimension of 47.0° and a vertical dimension of 43.0°. The viewing distance was 1.5 metres and was controlled by a chin rest. Eye movements were recorded using an infra-red limbus reflection device (Skalar, IRIS, Skalar Medical B.V.) with a linear range of ±20°, and an optimal resolution of 2 min arc and bandwidth DC to 100 Hz. The target stimulus was controlled by a modified REX (Real-Time Experimentation platform) data acquisition and analysis system developed for PC by Dr. T. Hain (Hain, 1995).

Procedure

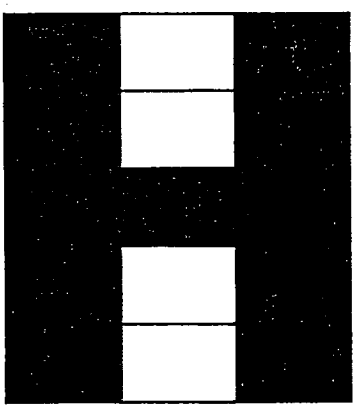
All participants read the information sheet and signed a consent form before commencing the experiment (Appendices 3 and 4 contain copies of the information sheet and consent form). PTSD Group participants were informed that the experiment did not contain any trauma-related material, but that trauma-related questionnaires were to be completed after the experiment.

Imaging Task Instructions: Practise trials for '0' / 'O', 'H', and '1' / 'L'

The imaging task was adapted from Logie, Zucco, and Baddeley (1990). Participants were shown a three by five matrix of white squares and told that some of the squares would be blackened to form an upper or lower case letter, from A to F, or

number, from one to nine, to be identified by the participant. Participants were told that taped instructions would inform them as to whether each square was to be black or white, starting at the top left square and proceeding down each column in turn.

Participants were next shown a matrix containing the character for the number '0' or the letter 'O' (Appendix 5 contains the letter and number characters). After participants correctly identified these characters, it was explained that if participants thought that a character could be both a number or letter, as in the example of number '0' and letter 'O', they were to simply choose one answer, as some of the test letter and number characters were identical and so both answers were correct. Participants were also told that they did not need to differentiate whether letter characters were upper or lower case in their answers. Next the 'black and white' instructions for the letter 'O' were spoken for participants to follow. Once participants understood the task, they were given a practise trial to complete on paper. This practise trial was the letter 'H', and participants were told the correct answer if they responded incorrectly.



BLACK, BLACK, BLACK, BLACK, BLACK

WHITE, WHITE, BLACK, WHITE, WHITE

BLACK, BLACK, BLACK, BLACK, BLACK

Figure 1. The imaging matrix and instructions for the letter H.

Participants were next informed that they would have to imagine the matrix and perform the visualisation task. Thus, they were given a practise trial to complete using visualisation. This trial answer was a lower case 'L', or number '1'- which was not repeated in the experiment, and again the participant's answer was corrected by the tester if necessary.

Instructions for Non-verbal Responses

Because the participants were instructed not to move their head during the measurement of EMs, the participants' responses in the imaging trials were given by hand signals. Number answers were signalled with the right hand. The participant was told to raise each finger, starting from the thumb, which was counted as '1', and to count each finger until the participant had reached the little finger,'5', leaving all fingers raised in between. The little finger became the sign for '6' when the other fingers were put down. The numbers '6' to '9' were indicated by raising and counting each finger, starting from the little finger, '6', and ending at the digit finger, '9', with the thumb remaining down. This procedure was repeated for the letter sequence on the left hand. The thumb indicated 'A', with each finger being raised to reach 'E', the little finger. The little finger became 'F' when the other fingers were put down. Participants were allowed to practise signing until they felt confident with the procedure. They were told to simply raise a fist if they did not know an answer during the experiment.

Experimental Procedures

Each participant was seated in an adjustable chair and was made as comfortable as possible, with his or her head in a chin rest. The room luminance level was 1.0 Lux. The experiment consisted of two baseline tasks, Baseline EMs and Baseline Imaging, and two experimental tasks, Imaging + Fixation and Imaging + EMs. The order of these four tasks was counterbalanced. Each of the three imaging tasks (Baseline Imaging, Imaging + Fixation, and Imaging + EMs) consisted of six trials of visualising the taped instructions, for constructing a letter or number character in an imaginary matrix, and responding with hand signals.

In the **Baseline Imaging Task** participants looked at the blank stimulus screen whilst they performed the imaging trials. The **Baseline EMs Task** involved participants performing rapid EMs to track a moving red target spot. These rapid EMs simulated the EMs elicited in EMDR, as judged by an independent EMDR trained therapist. Prior to EMs testing, the recording equipment was calibrated using 20 ° trials. During EMs testing the participants tracked the moving target spot as it travelled 44°/second, with a horizontal target stimulus distance of 20°. The EMs elicited during the Baseline EMs phase and Imaging + EMs Task were identical.

The **Imaging + EMs Task** involved participants performing the rapid EMs, whilst simultaneously completing the imaging trials. Participants were told that it was important for them to attend to the EMs component of the task, as well as to the imaging component of the task. Participants were able to rest their eyes between imaging trials.

In the **Imaging + Fixation Task** the participant was required to complete the imaging task whilst fixating on a stationary target spot, which was in the centre of the stimulus screen. Additionally, the target spot used in the Imaging + EMs and the

Baseline EMs tasks was moved horizontally through 20 ° and at a rate of 44°/second across the stimulus screen, and the participants were instructed not to track this second spot.

Self-Report Data Acquisition

Upon completion of the experiment, participants were asked to provide a verbal rating for how much subjective anxiety was provoked by each task (Baseline EMs, Baseline Imaging, Imaging + EMs, and Imaging + Fixation). This rating was on a scale of one to ten, where one was 'no anxiety' and ten was 'extreme anxiety'. Likewise, participants rated the difficulty of each of the four tasks, on a scale where one indicated 'no difficulty' and ten indicated 'extreme difficulty'.

Diagnostic Scales

Finally, participants were administered their relevant diagnostic scales and the Vocabulary scale of the WAIS-R (1981). The PTSD group participants were given the Posttraumatic Stress Diagnostic Scale (Foa, 1995) and then the Impact of Events Scale (Revised) (Weiss, 1993). All participants were given the SCL-90-R (Derogatis, 1993).

Design

The independent variable, Group, was either Control or PTSD. In the analysis of Groups' imaging ability, the dependent variable, Task, was Baseline Imaging, Imaging + Fixation, and Imaging + EMs, producing a mixed 2 (Group) X 3 (Task) design. In the analyses of Groups' subjective anxiety, difficulty ratings, and average heart rates, the dependent variable, Task, was Baseline EMs, Baseline Imaging,

Imaging + Fixation, and Imaging + EMs, producing a mixed 2 (Group) X 4 (Task) design. Descriptive and inferential statistical analyses (T-tests, ANOVAs, Tukey HSD post-hoc tests) were conducted where appropriate, and statistical significance was accepted at the 0.05 alpha level. The raw data can be found in Appendix 6.

Results

Group Characteristics

The descriptive statistics for Group characteristics are presented in Table 1. The group characteristics were not significantly different with regards to age, $t(24) = -.481$, $p > .05$; sex, $t(24) = .397$, $p > .05$; years of education, $t(24) = 1.149$, $p > .05$; and Vocabulary scores, $t(24) = 1.202$, $p > .05$. The PTSD Group scored significantly more than the Control Group on each of the SCL 90-R dimensions, $p \leq 0.001$. This demonstrated that the PTSD Group had significantly higher levels of distressing symptoms than the PTSD Group.

Table 1

Range and mean (SD) scores for the Control and PTSD Groups for age, sex, years of education, Vocabulary, and the SCL-90-R and Impact of Event (Revised) scales.

	PTSD Group			***Control Group***		
	Min	Max	Mean *(SD)*	Min	Max	Mean *(SD)*
Age	23	54	39.6 *(10.4)*	18	58	37.23 *(11.64)*
Years of Education	9	16	12.23 *(2.09)*	9	18	13.38 *(2.96)*
Vocabulary	10	19	13.2 *(3.02)*	10	19	14.62 *(3.18)*
Somatisation	46	81	65.9 *(11.05)*	30	56	45.15 *(7.31)*
Obsessive-Compulsive	60	81	70.85 *(7.79)*	30	68	50.69 *(11.56)*
Interpersonal Sensitivity	55	81	68.2 *(6.07)*	30	68	48.92 *(12.81)*
Depression	48	81	67.15 *(9.06)*	30	67	52.69 *(10.75)*
Anxiety	58	81	70.7 *(7.91)*	30	62	40.69 *(12.74)*
Hostility	49	81	64.4 *(10.82)*	30	65	40.46 *(12.61)*
Phobic Anxiety	30	81	67.2 *(14.05)*	30	61	34.23 *(10.43)*
Paranoid Ideation	60	79	66.5 *(5.41)*	30	69	41.31 *(15.57)*
Psychoticism	57	77	67 *(7.48)*	30	64	42.23 *(14.04)*
SCL-GSI	60	81	70.85 *(7.64)*	32	64	48 *(10.66)*
SCL-PST	56	79	68.69 *(6.43)*	33	64	49.15 *(9.86)*
SCL-PSDI	50	81	64.77 *(8.37)*	37	61	47.38 *(8.21)*
IMPACT of EVENT	28	84	49.7 *(13.89)*			
Intrusion	6	30	19.46 *(5.98)*			
Avoidance	8	32	15.69 *(6.47)*			
Hyper-arousal	7	22	14.54 *(5.03)*			

* The PTSD group contained 7 females and 6 males, and the Control group contained 8 females and 5 males.

Imaging Ability, Eye Movements, and Eye Fixation, in the PTSD and Control Groups

Analysis of Imaging Ability

The data were imaging scores for each participant in three Task conditions, Baseline Imaging, Imaging + Fixation, and Imaging + EMs. The data analysis involved an analysis of variance with one between factor (Groups: PTSD, Control) and one repeated measures factor (Task: Baseline Imaging, Imaging + Fixation, Imaging + EMs). Figure 2 shows the imaging ability of the two Groups as a function of the three tasks (Baseline Imaging, Imaging + Fixation, Imaging + EMs). The Groups main effect was non-significant, ($F(1,2) = 1.06$, $p > .05$). This demonstrated that there was no difference between the PTSD (53.24, $SD = 22.53$) and Control Groups (60.25, $SD = 22.41$) in mean imaging ability. The Group X Task interaction was non-significant, ($F(2,48) = 0.395$, $p > .05$).

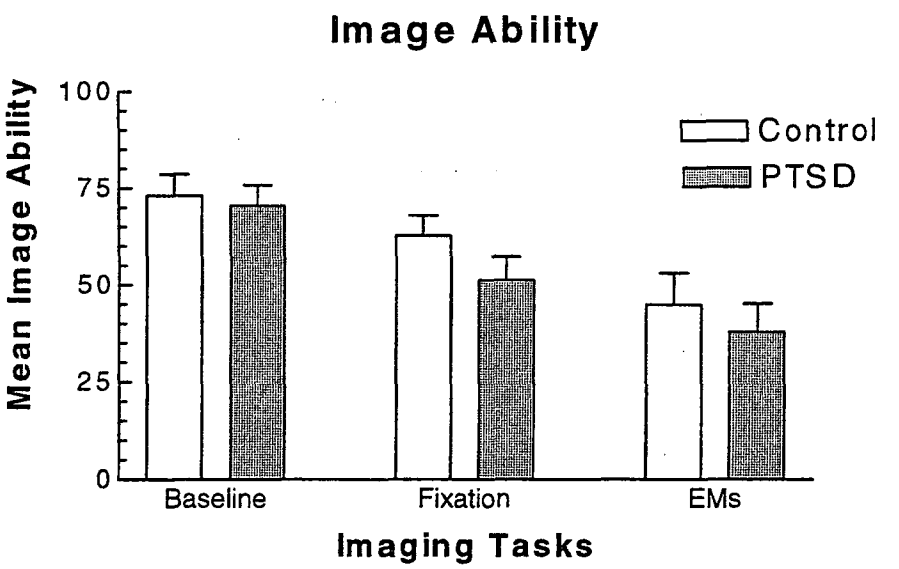


Figure 2. PTSD and Control Groups' mean *(St. Error)* imaging ability scores for Baseline Imaging, Imaging + Fixation, and Imaging + EMs.

Figure 3 shows imaging performance in the Baseline Imaging, Imaging + Fixation, and Imaging + EMs tasks, collapsed across groups. There was a highly significant Task main effect, ($F(2,48) = 18.205$, $p < .00001$), which shows that there were differences between the Baseline Imaging, Imaging + Fixation, and Imaging + EMs tasks. Tukey HSD post hoc analysis showed that imaging ability was highest in the Baseline Imaging task (71.79, *SD = 19.29)*, and that there was a significant reduction in imaging ability in the Imaging + Fixation task (57.05, *SD = 20.64)*, which in turn was followed by a significant reduction in imaging ability in the Imaging + EMs task (41.41, *SD = 27.37).*

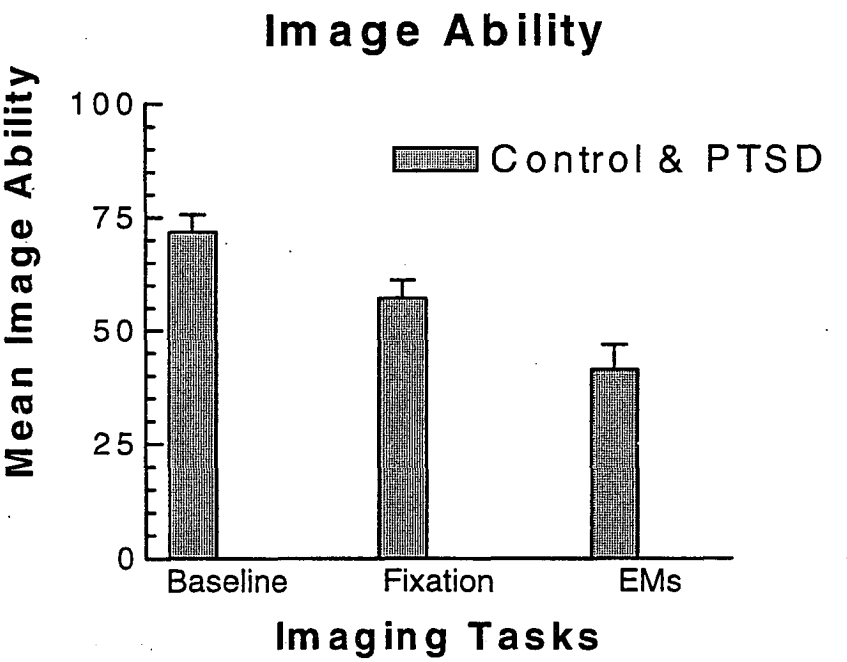


Figure 3. The combined Groups' mean *(St. Error)* imaging ability scores for Baseline Imaging, Imaging + Fixation, and Imaging + EMs.

Analysis of Eye Movements

Possible differences were examined in eye movement performances in the Baseline EMs task and in the six Imaging + EMs trials. The raw data in this analysis

were participants' scores for eye movement gain (eye movement velocity/ target movement velocity). Eye movement gain was averaged across the six experimental Imaging + EMs trials. An analysis of variance was conducted with one between factor (Groups) and one repeated measure (Gain: Baseline EMs and averaged eye movement gain in Imaging + EMs). The results showed that the Groups main effect was non-significant, ($F(1, 24) = 0.31$, $p > .05$), and demonstrated that there were no differences in eye movement gain between the PTSD and Control Groups. The Gain main effect was also non-significant, ($F(1, 24) = .09$, $p > .05$), and demonstrated that there were no significant differences in gain between the Baseline EMs and Imaging + EMs taskss. The Groups X Gain interaction was also non-significant, ($F(1, 24) = 0.006$, $p > .05$).

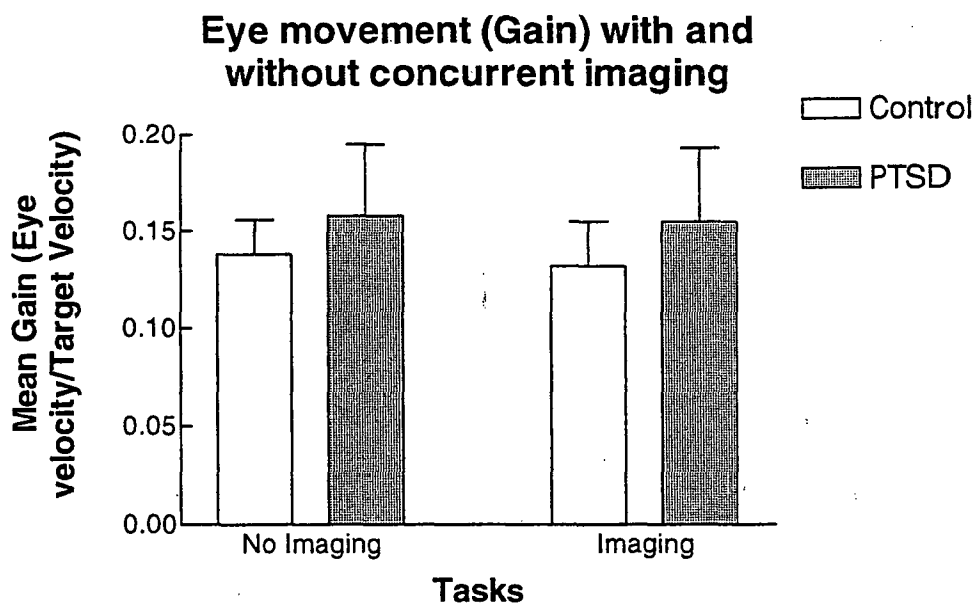


Figure 4. The mean (*St. Error*) Gain (Eye Velocity/Target Velocity) scores for the PTSD and Control Groups, measured during Baseline EMs and Imaging + EMs.

Analysis of Eye Fixation

An examination of the eye movement records in the Imaging + Fixation condition showed that eye blinks and occasional small saccadic eye movements

occurred, and their frequency were similar for participants in Control and PTSD Groups. Visual inspection of the eye fixation data showed that participants kept their eye on the stationary target fixation point.

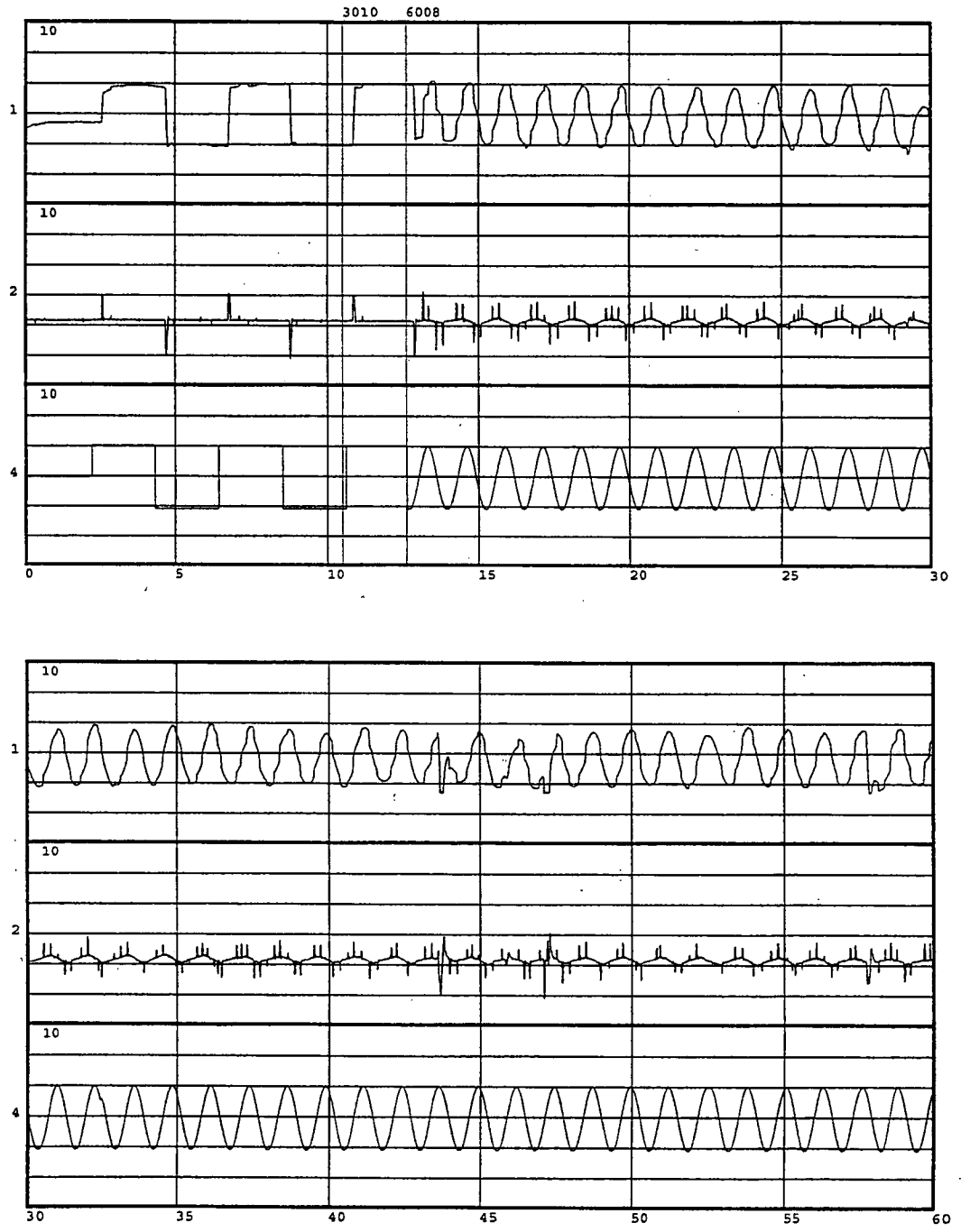


Figure 5. Recording of 60 seconds of eye movements during Baseline EMs task, showing the eye position signal in channel 1, eye velocity in channel 2, and target stimulus signal in channel 3, for Control participant: identification number 2.

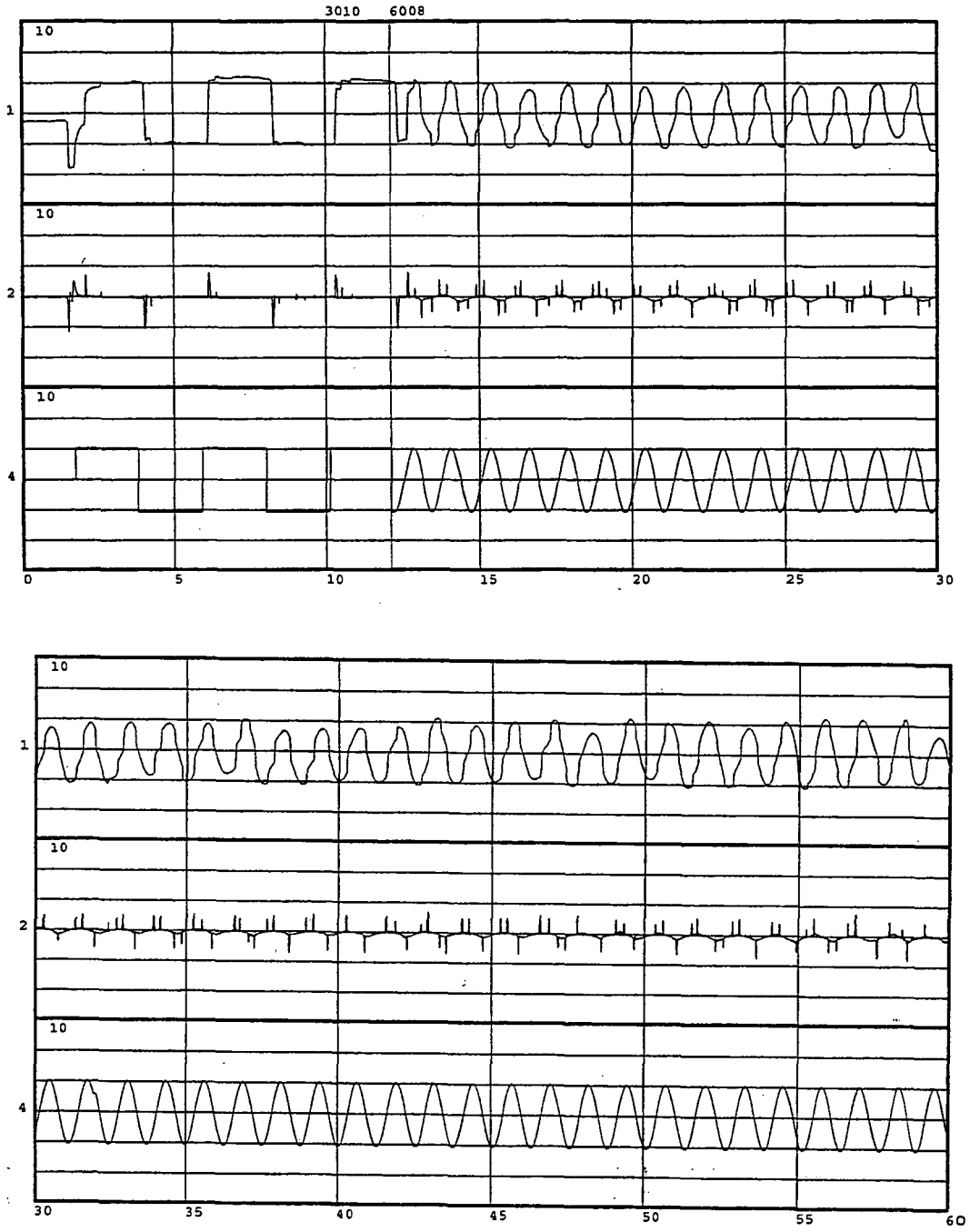


Figure 6. Recording of 60 seconds of eye movements during Imaging + EMs task, showing eye position signal in channel 1, eye velocity signal in channel 2, and target stimulus signal in channel 3, for Control participant: identification number 2.

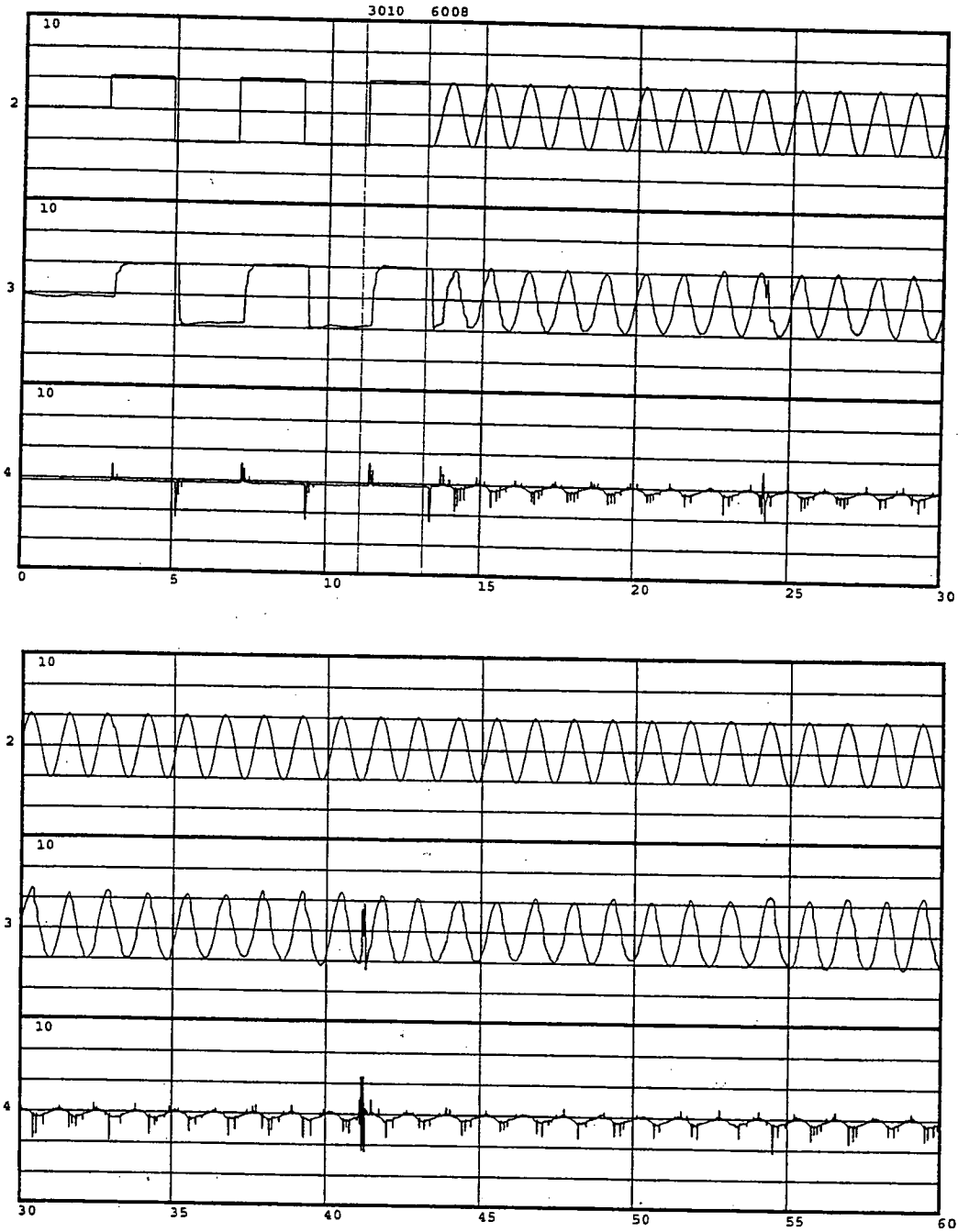


Figure 7. Recording of 60 seconds of eye movements during Baseline EMs task, showing the target stimulus signal in channel 1, eye position signal in channel 2, and the eye velocity signal in channel 3, for PTSD group participant: identification number 9.

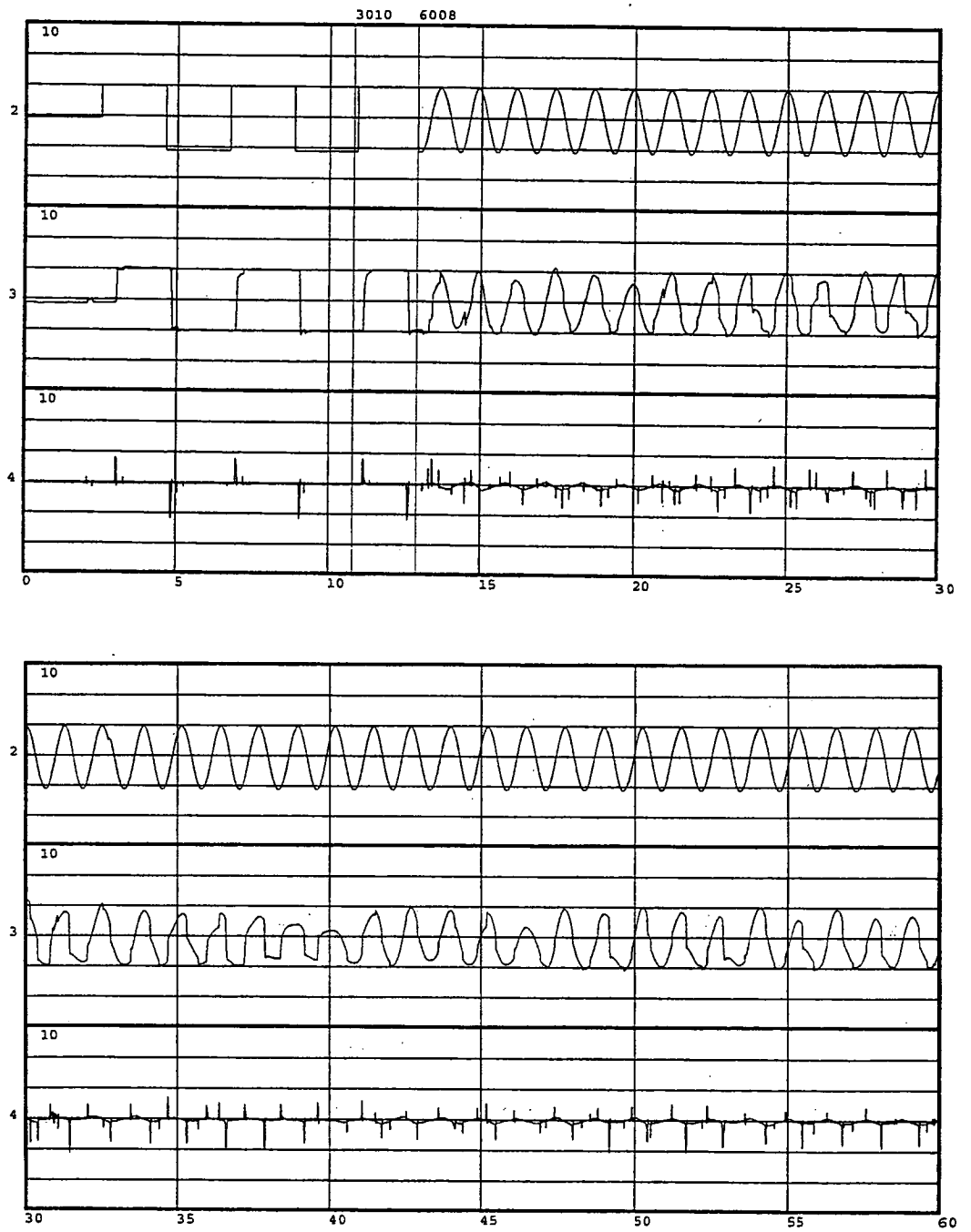


Figure 8. Recording of 60 seconds of eye movements during Imaging + EMs task, showing target stimulus signal in channel 1, eye position signal in channel 2, and eye velocity in channel 3, for PTSD group participant: identification number 9

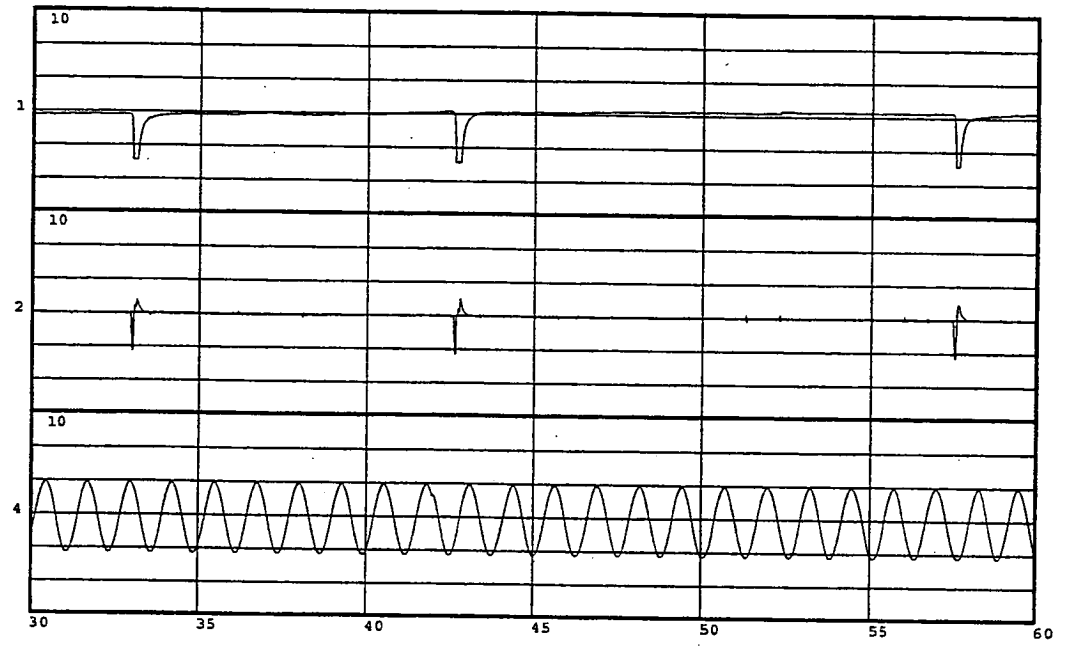


Figure 9. 30 seconds of eye fixation, with eye position signal in channel 1, eye velocity signal in channel 2, and moving stimulus signal in channel 3, for Imaging + Fixation, for Control participant: identification number 2.

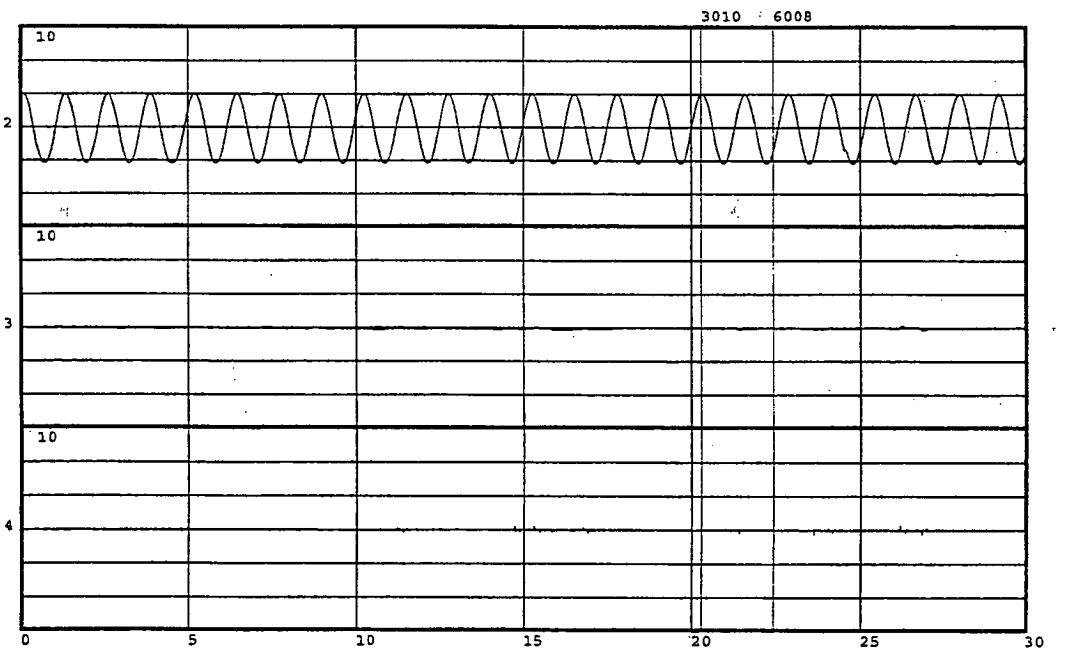


Figure 10. 30 seconds of eye fixation, with moving stimulus signal in channel 1, eye position signal in channel 2, and eye velocity signal in channel 3, for Imaging + Fixation, for PTSD Group participant: identification number 9.

Subjective Anxiety

Possible differences in subjective anxiety during Baseline EMs, Baseline Imaging, Imaging + Fixation, and Imaging + EMs, was also examined in the groups. The raw data were participants' ratings of subjective anxiety on a scale from 1, 'no anxiety', to 10, 'extreme anxiety', for each task. An analysis of variance was conducted on the anxiety scores with one between factor (Groups) and one repeated measure (Anxiety: Baseline EMs, Baseline Imaging, Imaging + Fixation, Imaging + EMs).

Table 2 shows the mean anxiety in the two groups. The analysis showed that the Groups main effect was highly significant, ($F(1, 24) = 21.69$, $p < .0001$), and showed that mean anxiety was significantly greater for the PTSD group. The Anxiety main effect was also significant, ($F(3,72) = 39.88$, $p < .0001$). Tukey HSD post hoc analysis showed that anxiety levels did not significantly differ for the Baseline EMs and Baseline Imaging tasks, and that these tasks were both significantly less anxiety provoking than Imaging + Fixation and Imaging + EMs. In turn, Imaging + Fixation provoked significantly less subjective anxiety than Imaging + EMs. There was no Group X Anxiety interaction, ($F(3, 72) = .43$, $p > .o5$).

Table 2

The range and mean (SD) subjective anxiety scores for the PTSD and Control Groups across Baseline EMs, Baseline Imaging, Imaging + Fixation, and Imaging + EMs.

	PTSD Group			*Control Group*		
	Min	Max	Mean *(SD)*	Min	Max	Mean (SD)
Baseline EMs	1	7	3.2 (*1.8*)	1	4	1.6 (*1.1*)
Baseline Imaging	1	6	3.9 (*1.7*)	1	4	2.3 (*1.3*)
Imaging + Fixation	3	10	6.3 (*1.9*)	1	6	3.9 (*1.3*)
Imaging + EMs	3	10	7.9 (*1.9*)	2	10	5.6 (*2.2*)

*13 participants in each group

Task Difficulty

Possible differences in the perceived difficulty of Baseline EMs, Baseline Imaging, Imaging + Fixation, and Imaging + EMs were examined in the groups. The raw data were participants' ratings of difficulty from 1, 'no difficulty', to 10, 'extreme difficulty', for each task. An analysis of variance was conducted on the difficulty scores with one between factor (Groups) and one repeated measure (Difficulty: Baseline EMs, Baseline Imaging, Imaging + Fixation, Imaging + EMs). Table 3 shows the mean difficulty ratings across the groups, for each task. The analysis showed that the Groups main effect was non-significant, ($F(1, 24) = 3.54$, $p > .05$), and so there were no differences in difficulty ratings between the PTSD and Control groups. The Difficulty main effect was significant, ($F(3, 72) = 47.49$, $p < .0001$), and demonstrated that Imaging + EMs was perceived to be the most difficult task followed

by Imaging + Fixation, Baseline Imaging, and Baseline EMs, respectively. There was no Group X Difficulty interaction, ($F(3, 72) = 1.186$, $p > .05$).

Table 3

The range and mean (SD) subjective difficulty scores for the combined Groups for Baseline EMs, Baseline Imaging, Imaging + Fixation, and Imaging + EMs.

Combined Groups' Subjective Difficulty Scores			
	Min	Max	Mean *(SD)*
Baseline EMs	1	8	2.92 *(2.15)*
Baseline Imaging	1	8	4.19 *(1.72)*
Imaging + Fixation	2	10	6.15 *(2.05)*
Imaging + EMs	2	10	7.69 *(2.29)*

*26 participants in combined groups

Pearson's 'r' correlations analysis showed that the combined Groups' subjective anxiety and perceived task difficulty ratings were significantly correlated for Baseline EMs (r = .49), for Baseline Imaging (r = .47), for Imaging + Fixation (r = .56), and for Imaging + EMs (r = .45).

Average Heart Rate Data

Average heart rate was measured during the baseline and experimental task conditions in the two groups. An analysis of variance was conducted to examine differences in average heart rate in the groups, with one between factor (Group) and one repeated measure (Average heart rate: Baseline EMs, Baseline Imaging, Imaging + Fixation, Imaging + EMs). Table 4 shows the mean heart rate during each task, for

the groups combined. The analysis showed that there was no Group main effect, ($F(1, 24) = .05$, $p > .05$), and so there were no differences in average heart rate between the PTSD and Control groups. The Average heart rate main effect was significant, ($F(3, 72) = 5.3$, $p < .005$), and demonstrated that average heart rate was significantly higher during the Imaging + EMs task, than during the other tasks. There was no Group X Average heart rate interaction, ($F(3, 72) = 1.13$, $p > .05$).

Table 4

The range and mean (SD) Average heart rate scores for the combined Groups for Baseline EMs, Baseline Imaging, Imaging + Fixation, and Imaging + EMs.

Combined Groups' Average Heart Rate Scores			
	Max	Min	Mean *(SD)*
Baseline EMs	53.9	100.5	76.8 *(11.5)*
Baseline Imaging	53.9	98.3	77.4 *(11.9)*
Imaging + Fixation	54.2	94.6	76.3 *(10.4)*
Imaging + EMs	56.1	101.8	80.9 *(11.3)*

*26 participants in combined groups

Discussion

The aim of this research was to investigate the kind of eye movements that are generated during EMDR treatment, and whether these eye movements have an effect on visual cognitive processing and anxiety as suggested by Shapiro (1995). The results showed the following: 1) The eye movements were mainly smooth pursuit eye

movement with occasional saccadic eye movement, in order to catch up when the moving target was faster than optimal pursuit eye movement. The results also showed that there were no significant differences in eye movement in the Baseline EMs task and the Imaging + EMs task or between the Control and PTSD groups.

2) The hypothesis that participants' imaging ability would be impaired in the Imaging + Fixation and Imaging + EMs tasks, compared to the Baseline Imaging task, was supported. Imaging ability decreased significantly more during Imaging +EMs, compared to Imaging + Fixation, as hypothesised. Participants reported that they were less able to concentrate on the imaging task when they had to keep their eyes on the moving stimulus.

3) Participants rated the Imaging + Fixation and Imaging + EMs tasks, as significantly more anxiety provoking and more difficult than the baseline tasks: Baseline Imaging and Baseline EMs. The Imaging + EMs task was rated as significantly more anxiety provoking and more difficult than the Imaging + Fixation task.

4) The difficulty of the Imaging + EMs task was reflected in the combined groups' increased average heart rate during this task. Thus, anxiety was increased in the Imaging + EMs task. Likewise, there was no significant reduction in the combined groups' mean average heart rate during the Baseline EMs task, which fails to support the proposal that EMs induce a 'compelled relaxation response'.

In summary, these findings fail to support Shapiro's (1995) evidence that the EMs facilitate cognitive processing and concentration, and by inference they fail to support her proposal that the EMs induce relaxation by causing physiological changes which activate the parasympathetic nervous system.

How may the finding that imaging ability was impaired in the Imaging + Fixation task be explained? In this task the participant was presented with a fixation

point to focus on whilst a spot cycled from left 10° to right 10° at a rate of 44°/second. Thus, the nature of the task resembled those studies in visual perception in which it has been shown that a visual stimulus briefly flashed or moved in peripheral vision has the capacity to inhibit visual processes at threshold in central vision (Breitmeyer, Valberg, Kurtenbach, & Neumeyer, 1980; Breitmeyer, 1980; Breitmeyer & Ganz, 1976). These interactions have been explained in terms of visual masking which refers to a reduction in visibility or suppression of a target stimulus by a spatially overlapping or adjacent masking stimulus which is in close temporal proximity to the target (Breitmeyer & Ganz, 1967).

The nature of the Imaging + Fixation task also bears a close resemblance to neurophysiological studies which involve interactions between central and peripheral visual areas at the single cell level and have variously been referred to as the 'periphery' (McIllwain, 1966), 'shift' (Kruger, 1980), or the 'far out jerk' effect (Breitmeyer et al., 1980). The present results are similar to the above studies and, in addition, show that a moving stimulus also has the ability to impair a visual cognitive process. The 'far-out jerk effect' has been explained in terms of the activity within a particular pathway in the visual system called the magnocellular (M) pathway. Research has shown that the M pathway plays a major role in the direction of visual attention and in the programming of eye movement (Lennie, 1993). Lennie (1993) argues that the M pathway provides the signal which facilitates a saccadic eye movement for detecting objects in peripheral vision, and that this is under the control of attention. The proposal by Lennie (1993) is also consistent with Posner and Petersen's (1990) model of attention which involves an anterior attention system and a posterior attention system. It is proposed that orientation to a visual location is performed by the posterior attention system which lies in the dorsal visual pathway

that has its primary cortical projection area in V1 and extends to the parietal lobe. The dorsal pathway primarily contains the M cells and is involved in shifting gaze.

Thus, it seems that the significant reduction in imaging ability may be explained in terms of a capturing of visual attention by the moving stimulus, or an inhibitory process initiated by a moving peripheral stimulus which decreases central cognitive processes, or simply as the result of distraction. It is important to note, however, that the moving target stimulus did not cause smooth pursuit eye movement to occur as a consequence and only very small and occasional saccadic eye movements were made, as shown by the eye fixation records.

The results show that performance of EMs in response to a moving target, in the Imaging + EMs task, was also an interference which prevented participants from constructing a visual image. *How may this finding that imaging ability was impaired in the Imaging + EMs task be explained?* As above, this result can be explained in terms of activation of the posterior attention system which may reduce the attention resources allocated to the imaging task, a 'shift effect' suppressing central cognitive processes needed for imaging, or simply by a distraction effect from performing EMs. The finding that imaging ability is decreased in the Imaging + EMs task also supports Dyck's (1993) theory that the EMs serve to distract EMDR clients from visualising their traumatic memories. It is proposed that this distraction effect is explained by a limited capacity of the Visual Spatial Sketchpad (VSSP) of the working memory (Andrade, et al., 1997). The imaging task in this experiment, like the imaging of traumatic memories, is proposed to require the processing resources of this VSSP. However the performance of eye movements, which increase visual input from the environment, may deplete the processing resources of the VSSP, and so may disrupt concurrent visualisation (Andrade, et al., 1997).

The Imaging + EMs task was rated the most anxiety provoking and difficult task and, consistent with these ratings, participants' average heart rates were significantly elevated during this task, compared to the Baseline Imaging, Baseline EMs and Imaging + Fixation tasks. Moreover the Baseline EMs task did not reduce participants' average heart rates. *How can this failure to elicit a 'compelled relaxation response' to performing rapid EMs, as found by Wilson, et al. (1996), be explained?* The answer may simply be that participants were motivated to score well on the Imaging + EMs task and so may have become more anxious when they could not perform the necessary mental operations for the imaging task.

Alternatively, in EMDR, clients may not be motivated to visualise their trauma, due to fear and habitual avoidance, and so may be relieved when they can not visualise their trauma or concentrate on trauma-related beliefs at the same time as performing rapid EMs. This may be why some clients become relaxed during EMDR, rather than because rapid eye movements directly cause physiological changes which decrease psychophysiological arousal. The present results are consistent with the view that EMs may reduce clients' fear and anxiety in EMDR because the vividness of the traumatic imagery is decreased (Andrade, et al, 1997). Thus, feelings of relief and relaxation may be repeatedly paired with the traumatic image, extinguishing the client's anxiety response (Dyck, 1993). This may in turn increase the client's sense of mastery over the traumatic memory, and his or her acceptance of the cognitive and behavioural therapeutic procedural elements of EMDR. Thus, EMDR may have a role in the treatment of PTSD when traditional exposure therapies have failed or are inappropriate because clients are too anxious or avoiding to cope with a standard exposure treatment (Andrade, et al., 1997), such as flooding which has been reported to be an anxiety provoking and aversive exposure treatment (Scott & Stradling, 1997;

Solomon, Gerrity, & Muff, 1992). The EMs may facilitate the treatment effects of EMDR by distracting clients from their anxiety, thus making EMDR a more tolerable exposure therapy.

Future research needs to investigate the separate effects of saccadic and smooth pursuit eye movements on imaging ability and on the treatment outcomes of EMDR. Thus the imaging task could be performed whilst the participant focuses on a target spot alternately flashing in the left and right visual fields (eliciting saccadic EMs), or whilst the participant tracks a slow moving object (eliciting smooth pursuit EMs). Based on the conclusion that slow EMD did not impair participants' emotional processing of an aversive photograph, in Tallis and Smith's (1993) study, because visualisation of the aversive photograph was not impaired, it is suggested that saccadic EMs may impair imaging ability more than slow, smooth pursuit EMs. This would support Irwin and Carlson-Radvansky's (1996) proposal that saccadic EMs suppress cognitive processing. Also whether or not participants in EMDR exhibit a 'compelled relaxation response' needs to be confirmed by controlled research.

EMDR is a treatment for PTSD which relies on ambiguous, physiological base theories regarding the effects of rapid eye movements on information processing and psychophysiological arousal. It has been claimed that the credibility of EMDR is undermined by this "sketchy neurobiological theorizing" (Allen & Lewis, 1996, p 250), and without a sound rationale for each of the component parts of EMDR it is more difficult for therapists to be able to adapt this treatment to clinical practice. It is suggested here that rapid eye movements do not facilitate cognitive processing and do not induce relaxation, but may distract clients from their usual anxiety response to traumatic stimuli. This study therefore contributes to the understanding of the effects of EMDR. However more research is needed to investigate the underlying

mechanisms of EMDR, if therapists are to understand which components of EMDR result in positive treatment outcomes. This knowledge may lead to further refinement of the treatment procedure.

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Appendix 1. Newspaper advertisement

Post Traumatic Stress Disorder Study

A study is being conducted into how eye movements can be used to treat Post Traumatic Stress Disorder. Volunteers with Post Traumatic Stress Disorder are needed to spend two hours at the University of Tasmania, Hobart campus, performing eye movements and completing questionnaires. If interested please ring Jilli Fraser, 03 62 250 749.

Appendix 2. Poster for Community Mental Health Services

VOLUNTEERS WANTED

Have you been in a traumatic incident and now feel stressed and anxious?

Do you have Post Traumatic Stress Disorder?

People interested in participating in a Clinical Psychology Masters study are needed.

The study is an investigation of how eye movements can influence cognitive visual processing. Thus we need to monitor participants' eye movements for a short period of time (approx. 1 hour + questionnaire time) in the Psychology department of the Hobart University.

The aim of this study is to improve treatment strategies for people who are experiencing the negative effects of a traumatic event. Therefore participants who have been in a traumatic incident, ie. a car accident, as well as control participants are required.

If you are interested in participating please contact Jilli Fraser (03 62 250749) or Walter Slaghuis (03 62 262051)

Appendix 3. Information Sheet

Information Sheet

Title of Investigation: An assessment of the effect of eye movements on imaging in EMDR and treatment for PTSD

Chief Investigators: Dr Walter Slaghuis and Dr Montgomery

Researcher: Miss Jilli Fraser

Purpose of the Study: This study aims to test Shapiro's theory that the rapid eye movements elicited in her Eye Movement Desensitisation and Reprocessing (EMDR) treatment of Post Traumatic Stress Disorder (PTSD) facilitate cognitive visual processing. This study aims to add significantly to recent research concerning the theoretical components of EMDR.

As part of this research we require the assistance of people suffering from PTSD or people who have been in a traumatic incident which has caused stress in their lives, and people who do not have PTSD. There is no payment for participation and the study does not provide treatment for PTSD. Participants are not required to talk about their traumatic memories to the researchers. The results of volunteers' participation will help explain recent reports on the effectiveness of EMDR in treating PTSD.

If you decide to participate in the study, your task will be to track a red light, moving rapidly across a screen, in a dark room with the researcher and chief investigator. Thus, we will require that you wear a comfortable helmet which is attached to equipment which records your eye movements. Also at different stages you will be asked to visualise a number from 1 to 9 or a letter from A to F, according to taped instructions. Practise and explanation of these tasks will be provided before commencement of the study. The eye tracking and number/letter visualisation tasks will be performed separately and simultaneously.

You will also be asked to fill out the SCL - 90-R (Derogatis, 1993), a questionnaire concerning mental health symptomatology, and the Posttraumatic Stress Diagnostic Scale (Foa, 1995). Also you will be given short visual analogue scales concerning how easy/difficult you found each task, and how relaxing/anxiety-provoking you found each stage of the study. We will also measure your heart rate as an indication of relaxation, and so you will be required to an ear clip, which is a very safe procedure that will be explained in full at the time of the study.

The information that you give us will be kept in the strictest confidentiality. Only the researchers conducting the investigation will have access to the identifying data. The results of the study will be available on request.

Participation is entirely voluntary. You are permitted to discontinue involvement in the study at any stage without prejudice from the researcher or chief investigator. If you require any further information at any stage please contact Dr Walter Slaghuis on 62 262 237 or Jilli Fraser on 62 250 749. If you have any ethical concerns or complaints about the manner in which the project is being conducted, you may contact the following member of the University of Tasmania Ethics Committee:

Mrs Chris Hooper : 62262763

This study has been approved by the University of Tasmania Ethics Committee and complies with the laws of the state. Should you require assistance in coping with PTSD staff is available to assist with an appropriate referral. You will be given copies of the information sheet and consent forms to keep. **Thankyou for your participation.**

Appendix 4. Consent Form

Consent Form

I _____ consent to participate in the study being conducted by Dr Walter Slaghuis, Dr Montgomery, and Miss Jilli Fraser. I have been informed that the study is being conducted in an attempt to understand the effects of eye movements on visual cognitive processing ability. I understand that I will be required to wear equipment which will allow my eye movements to be recorded and my heart rate to be monitored. I also understand that I will be asked to complete a questionnaire concerning mental health symptomatology. I have been informed that all information is confidential, and that I can discuss my results with the investigators.

I understand that I may withdraw from the study at any time by stating a wish to do so. I also understand that if I have any concerns about the study I may discuss them with the investigators Dr Walter Slaghuis or Jilli Fraser on (03) 62 250749.

I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this study and understand that I may withdraw at any time. I agree that research data gathered for the study may be published provided that I cannot be identified as a participant.

Signature of participant:

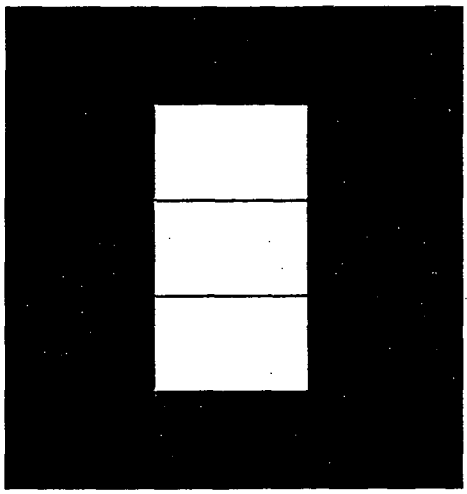
Date:

I have explained this project and the implications of participation in this project to this volunteer. I believe that the consent is informed and that he/she understands the implications of participation.

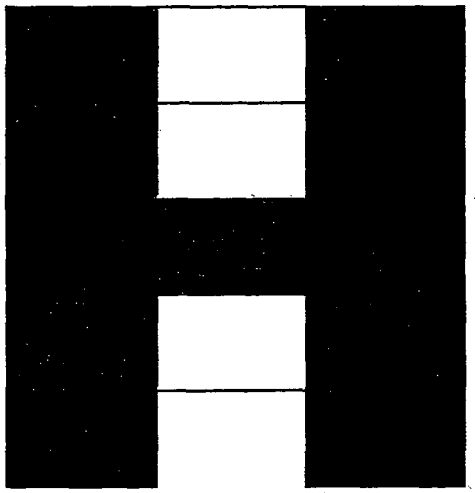
Signature of Researcher:

Date:

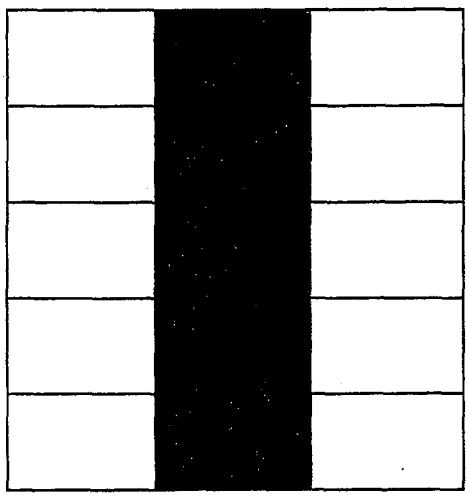
Appendix 5. Letter and number characteristics in the Imaging Task



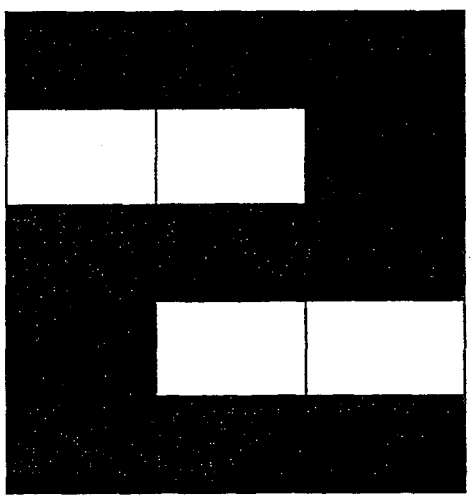
Example Matrix; "O" or "0"



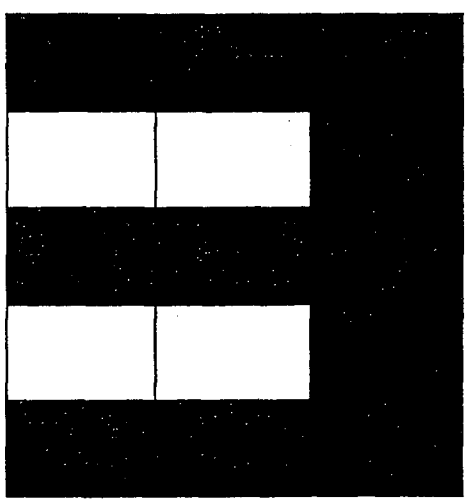
Practise Trail 1; The letter "H"



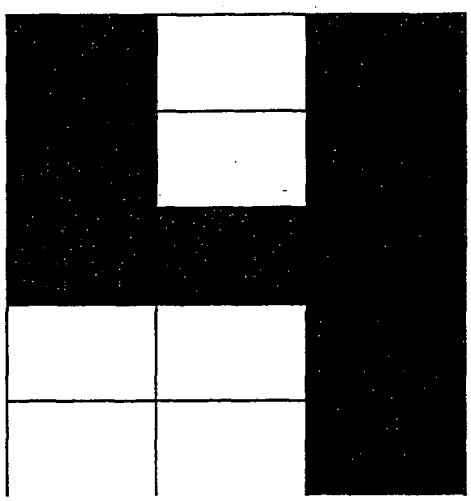
Practise Trial 2; "I" or "1"



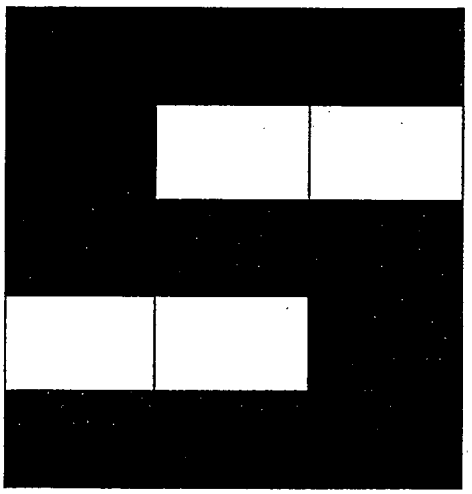
The number "2"



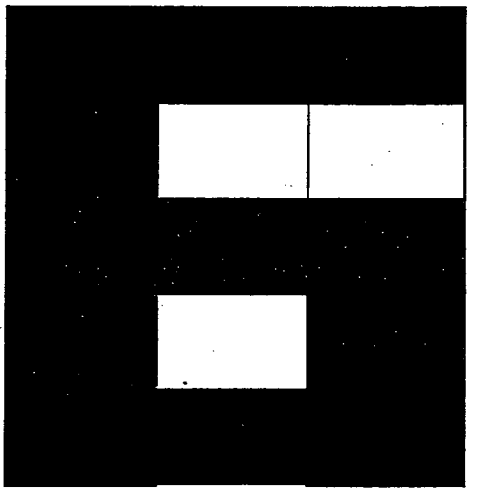
The number "3"



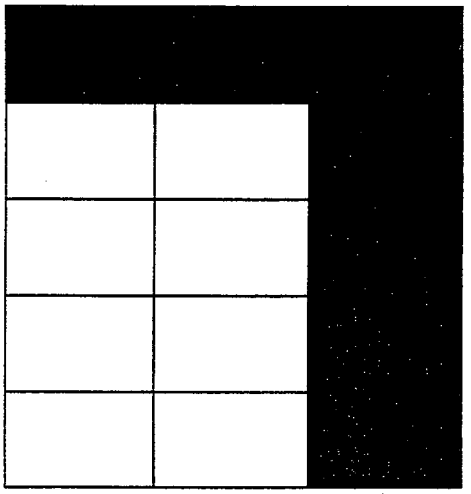
The number "4"



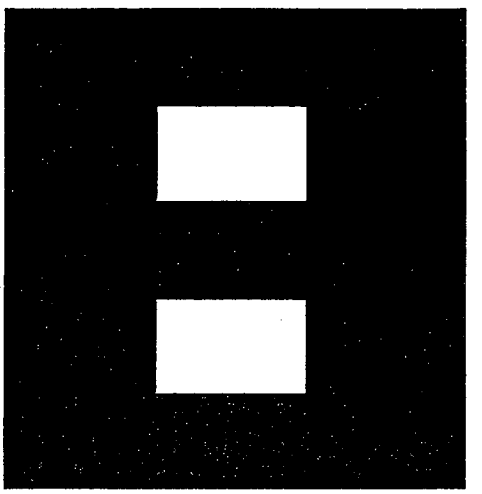
The number "5"



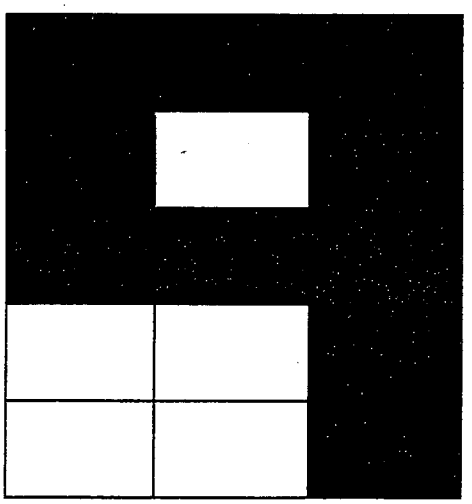
The number "6"



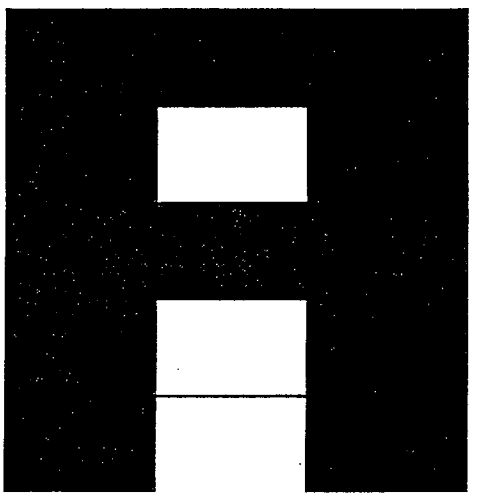
The number "7"



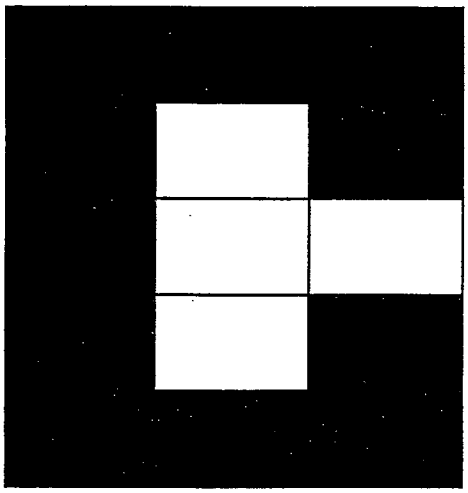
The number "8" or letter "B"



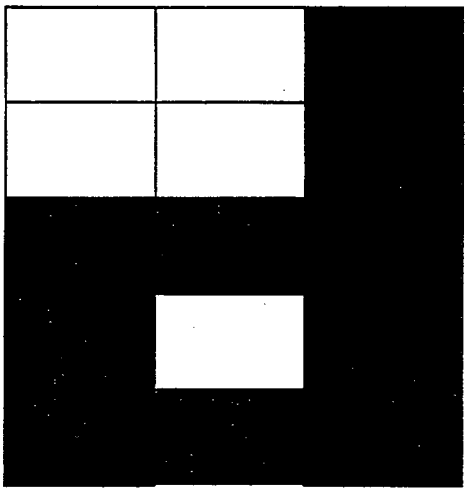
The number "9"



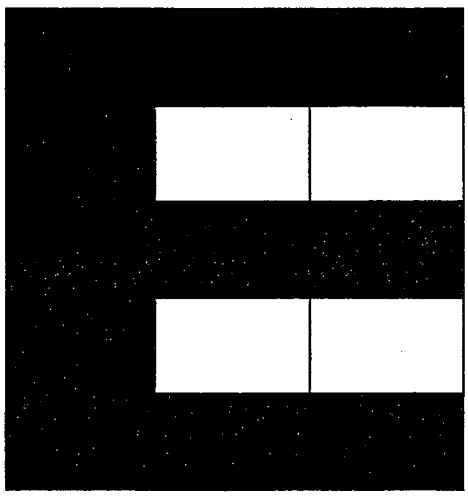
The letter "A"



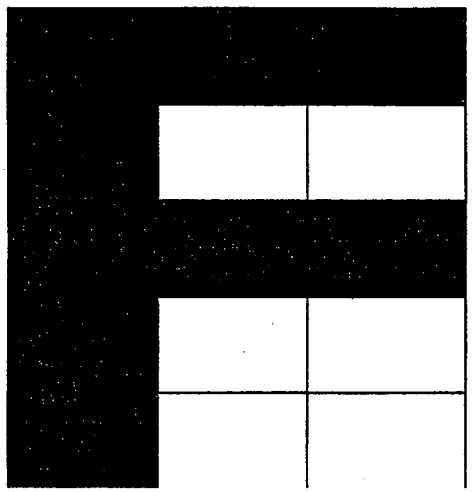
The letter "C"



The letter "d"



The letter "E"



The letter "F"

Instructions for the letter "O" or number "0";

BLACK, BLACK, BLACK, BLACK, BLACK (pause)
BLACK, WHITE, WHITE, WHITE, BLACK (pause)
BLACK, BLACK, BLACK, BLACK, BLACK.

Instructions for the letter "l" or number "1";

WHITE, WHITE, WHITE, WHITE, WHITE (pause)
BLACK, BLACK, BLACK, BLACK, BLACK (pause)
WHITE, WHITE, WHITE, WHITE, WHITE.

Instructions for the number "2';

BLACK, WHITE, BLACK, BLACK, BLACK (pause)
BLACK, WHITE, BLACK, WHITE, BLACK (pause)
BLACK, BLACK, BLACK, WHITE, BLACK

Appendix 6. Raw Data

Table 1.

The raw data for each participant in the Control Group, for each of the variables.

Participants in the Control Group, numbered 1-13													
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**
Age	18	38	33	24	24	37	39	45	40	42	58	56	34
Sex	1	1	2	2	1	2	2	2	2	2	2	1	2
years of education	13	10	11	18	16	13	13	14	13	18	9	10	16
Vocabulary	18	11	10	19	17	12	14	15	11	12	18	15	18
Somatisation	18	11	10	19	17	12	14	15	11	12	18	15	18
Obsessive-Compulsive	55	52	58	30	46	60	66	68	30	50	50	46	48
Interpersonal Sensitivity	49	30	65	30	49	58	68	62	30	52	46	49	48
Depression	60	45	59	45	49	65	67	63	41	52	59	30	50
Anxiety	48	30	57	44	30	52	62	56	30	30	30	30	30
Hostility	30	30	57	30	50	49	65	30	30	46	49	30	30
Phobic Anxiety	30	30	61	30	30	54	30	30	30	30	30	30	30
Paranoid Ideation	30	30	54	30	49	69	62	63	30	30	30	30	30
Psychoticism	58	30	53	30	30	64	53	58	30	53	30	30	30
SCL-GSI	52	45	60	32	41	62	64	61	37	47	41	41	41
SCL-PST	52	44	61	33	44	64	64	58	39	48	44	43	45
SCL-PSDI	50	48	52	37	40	54	59	61	37	49	42	50	37
Impact of Event	0	0	0	0	0	0	0	0	0	0	0	0	0
Intrusion	0	0	0	0	0	0	0	0	0	0	0	0	0
Avoidance	0	0	0	0	0	0	0	0	0	0	0	0	0
Hyper-arousal	0	0	0	0	0	0	0	0	0	0	0	0	0
Baseline Gain	.15	.14	.27	.10	.09	.07	.26	.08	.08	.14	.14	.19	.09
Average Gain	.14	.17	.23	.05	.05	.04	.29	.08	.08	.08	.14	.25	.11
Imaging + EMs	83.3	66.7	33.3	16.7	83.3	33.3	16.7	0	83.3	16.7	33.3	50	66.7
Imaging + Fixation	66.7	50	33.3	83.3	83.3	50	66.7	66.7	83.3	33.3	50	66.7	83.3
Baseline Imaging	83.3	66.7	100	50	100	50	83.3	66.7	66.7	50	50	83.3	100
Baseline EMs Anxiety	3	1	1	0	3	1	2	1	2	1	4	1	1
Imaging + EMs Anxiety	2	7	7	7	3	4	8	10	6	4	6	5	4
Imaging + Fixation Anxiety	1	6	2	4	4	4	5	5	4	4	5	3	4

Continued from the previous page.													
Participants in the Control Group, numbered 1-13													
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**
Baseline Imaging Anxiety	1	3	1	4	2	1	3	4	3	1	4	1	3
Baseline EMs Difficulty	7	1	1	4	1	1	1	1	2	1	4	2	1
Imaging + EMs Difficulty	4	9	7	10	3	8	8	9	7	8	7	7	7
Imaging + Fixation Difficulty	4	6	2	6	3	4	7	6	5	9	6	7	5
Baseline Imaging Difficulty	3	3	2	6	2	3	5	5	7	3	5	5	4
Baseline EMs Average Heart Rate	71.2	68.3	100	70.8	60.8	74.6	74.9	58.3	82.99	81.6	83.7	75.85	71.77
Imaging + EMs Average Heart Rate	68.44	79.1	101.8	77.14	70.5	80.6	79.56	64.8	99.1	85.95	86.3	84.4	79.7
Imaging + Fixation Average Heart Rate	67.1	75.05	83.1	69.5	75.5	75.3	67.61	60.8	88.5	78.2	91.9	82.7	77
Baseline Imaging Average Heart Rate	62.17	84.25	82.83	70.11	74.91	71.42	67.32	57.78	79.53	90.84	95.82	83.2	78.14

Table 2.

The raw data for each participant in the PTSD Group, for each of the variables.

Participants in the PTSD Group, numbered 14-26													
	14	**15**	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**
Age	23	35	28	53	46	36	54	36	29	46	49	31	49
Sex	1	2	2	2	1	1	1	2	2	1	2	2	2
years of education	13	16	15	12	10	13	9	13	14	12	12	10	10
Vocabulary	12	19	12	18	12	11	10	16	14	12	15	10	10
Somatisation	59	63	79	76	80	54	66	56	65	72	81	60	46
Obsessive-Compulsive	60	80	71	73	80	61	72	66	68	81	81	62	66
Interpersonal Sensitivity	66	63	66	70	73	67	70	70	69	81	72	64	55
Depression	67	63	66	75	72	67	77	69	54	81	72	62	48
Anxiety	71	67	71	72	81	64	80	63	65	81	81	65	58
Hostility	66	75	57	63	80	50	63	49	68	81	74	57	54
Phobic Anxiety	69	63	63	54	74	66	80	67	80	81	81	65	30
Paranoid Ideation	62	69	65	72	79	60	60	71	66	65	69	62	65
Psychoticism	59	62	68	68	76	64	75	71	60	76	77	57	58
SCL-GSI	65	69	70	73	81	63	80	67	68	81	81	63	60
SCL-PST	63	65	72	71	79	64	74	69	64	77	73	66	56
SCL-PSDI	63	69	62	69	67	50	67	58	67	74	81	52	63
Impact of Event	36	52	47	55	61	36	43	51	57	51	84	28	45
Intrusion	17	23	20	21	20	12	20	19	18	20	30	6	27
Avoidance	12	10	18	13	23	11	13	17	19	17	32	11	8
Hyper-arousal	7	19	9	21	18	13	10	15	20	14	22	11	10
Baseline Gain	.10	.20	.27	.08	.11	.41	.01	.21	.22	.15	-.1	.12	.30
Average Gain	.20	.20	.20	.03	.08	.43	-.02	.16	.4	.11	.06	.13	.03
Imaging + EMs	66.7	50	33.3	33.3	33.3	16.7	0	33.3	33.3	60	16.7	100	16.7
Imaging + Fixation	66.7	50	33.3	83.3	83.3	50	66.7	66.7	83.3	33.3	50	66.7	83.3
Baseline Imaging	83.3	100	83.3	66.7	50	83.3	33.3	83.3	83.3	50	50	66.7	83.3
Baseline EMs Anxiety	2	1	1	3	4	7	5	2	1	3	4	5	4
Imaging + EMs Anxiety	7	3	6	7	8	8	10	10	9	8	10	7	7
Imaging + Fixation Anxiety	3	8	5	5	4	6	7	6	9	6	10	6	7

Continued on the next page.

Participants in the PTSD Group, numbered 14-26/													
	14	**15**	**16**	**17**	**18**	**19**	**20**	**21**	**22**	**23**	**24**	**25**	**26**
Baseline Imaging Anxiety	1	3	1	4	2	1	3	4	3	1	4	1	3
Baseline EMs Difficulty	7	1	1	4	1	1	1	1	2	1	4	2	1
Imaging + EMs Difficulty	2	10	9	9	9	8	10	10	9	8	10	3	9
Imaging + Fixation Difficulty	4	8	8	7	9	4	9	6	10	6	8	4	7
Baseline Imaging Difficulty	1	4	6	7	5	3	8	2	4	4	4	3	5
Baseline EMs Average Heart Rate	90.36	60.9	92.45	71.94	73.71	82.6	90.9	78.13	91.05	53.86	68	82.3	85.59
Imaging + EMs Average Heart Rate	95.72	63.05	93.58	75.36	75.53	84	92.29	76.99	89.76	56.08	70.42	87.93	85.65
Imaging + Fixation Average Heart Rate	94.64	63.62	91.41	70.1	75.87	73.6	89.3	68.1	84.36	54.18	66.9	87.71	72.72
Baseline Imaging Average Heart Rate	98.34	66.72	90.24	69.07	72.24	70.5	92.8	65.66	88.03	53.88	76	92.38	76.99